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HEIGHTS AND WEIGHTS OF WHITE AMERICANS*

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DATA on the height and weight of a national population or of particular segments of it serve many biological purposes.

1) They may reflect changes in body size and form within a country, and they permit comparisons among population groups. The relative contributions of heredity and environment to such differences, the health status of various populations or subgroups, and the desirability or effect of specific medical and public health programs can be thereby assessed.

2) As reference standards, national height and weight figures provide a rough estimate of an individual's or a group's relation to the norm in respect to growth, development, nutriture, and aspects of body size and form associated with or resulting from disease.

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3) Body size data are useful to designers of all kinds of equipment, whether industrial, military, or medical. The role of human dimensions in the design of equipment for safe, efficient, and comfortable use has long been acknowledged, if all too seldom apparent.

4) Various physiological processes involve body size, volume, or surface area, which are readily calculated from height and weight. Oxygen consumption, as in basal metabolism, maximum breathing and vital capacities, heat production and water loss, nutritional and replacement requirements, drug dosage and effects, absorption and effect of heat, light, and radiation—all these and many more come to mind.

The present attempt to synthesize available data into valid national figures stems primarily from our own research needs in the area of man-machine integration as a factor in accidental trauma, a major medical and public health problem. A further stimulus is the widespread concern over radiation exposure in population groups (*cf.* Lisco, 1949). While the values submitted here are by no means definitive, we believe they will not differ greatly from the true figures, whenever the latter are established. Until then, the need remains great, and a reasonable approximation should prove useful.

SOURCES OF DATA

There are no completely satisfactory data on height and weight for the American population. By "completely satisfactory" is meant a sizeable representative sample, measured recently by trained observers under standardized conditions, preferably with the subjects unclothed. It is hoped that the National Health Examination Survey of the United States Public Health Service, begun in October, 1959, will eventually furnish such information. Some 6,000 persons, carefully selected to represent the United States population between the ages of 18 and 79, will be examined. The results will not be at hand, however, for at least 3 years. In 1956, a model survey (Pett and Ogilvie, 1956) of the Canadian population, based on 22,000 subjects, aged 2 to 65-plus years (6308 persons over age 18), was published. The Canadians, even excluding the generally smaller inhabitants of Quebec (Pett and Ogilvie, 1957), are somewhat shorter and lighter than most American groups at corresponding ages.

In the United States, the recent monumental report by the Society of Actuaries (1959), though fulfilling its intended purposes of delineating

time trends and of relating physique, blood pressure, and mortality in broad general terms, must be deemed unsatisfactory by the above criteria. Based on nearly 5 million applicants for life insurance, the study may under-represent the lower socio-economic groups. The subjects, whose income level, race, and geographic distribution were not stated, were measured by a variety of uncontrolled techniques—indeed, 20% of the weights and 10% of the heights were admittedly not measured at all. Indoor clothing and shoes were worn, so that interpretation of the results depends heavily on speculation concerning clothing habits.

Presently available data on height and weight vary in amount and quality, being better for some segments of the American population than for others. Children, young men of military age, and white women between ages 18 and 70 have been fairly well investigated, whereas middle-aged and older men, as well as Negroes of both sexes, are less adequately described. In fact, studies on adult Negroes are so sparse as to preclude valid summary at present.

In the preparation of table 1, most of the major published studies since the mid-1930's were examined with respect to recency, sample size and composition, and anthropometric technique. Doubtless some research has been overlooked. Data from before World War II are still valid for birth size, which seems to have remained constant over the period, as well as for certain groups where other considerations, such as ancestry or socio-economic status, outweigh the well-established trend to increased size. In view of the limitations inherent in any one study, several samples were sought at all ages, but the number of samples dwindled sharply after the third decade. Accordingly, two of our own published series (Damon and McFarland, 1955; Damon, 1957) have been re-analyzed by age, and some unpublished investigations by others (Equitable Life Assurance Society, 1940; Gibbons *et al.*, 1953; Tyroler, 1958) as well as ourselves (Damon, 1954, 1960; Damon and Stoudt, 1958, 1959) have been employed. Of the many sources and compilations consulted, those actually used are appended, grouped by broad age spans.

METHODS

Each entry in table 1 represents a balancing of several factors among the sources available: date of survey, adequacy of measuring technique, number of subjects, and their selection in terms of ancestry, geography, health, and socio-economic status. The procedure can be illustrated by the height of 20-24 year old males, given as 68.7 inches, with a standard

TABLE 1

Heights and weights of White Americans

	MALES				FEMALES			
	HEIGHT (IN.)		WEIGHT (LBS.)		HEIGHT (IN.)		WEIGHT (LBS.)	
AGE	MEAN	S. D.	MEAN	S. D.	MEAN	S. D.	MEAN	S. D.
Birth	20.0	1.0	7.6	1.3	19.7	1.0	7.5	1.1
0-1 month	20.7	1.0	8.5	1.3	20.4	1.0	8.2	1.1
1-2	21.5	1.0	9.9	1.6	21.3	1.0	9.4	1.2
2-3	23.0	1.0	12.1	1.7	22.5	1.0	11.2	1.3
3-4	24.0	1.0	13.9	1.9	23.6	1.0	12.8	1.5
4-5	25.2	1.0	15.4	1.8	24.6	1.0	14.2	1.6
5-6	26.0	1.0	16.8	2.0	25.4	1.0	15.6	1.8
6-7	26.6	1.0	18.0	2.1	26.1	1.0	17.0	1.9
7-8	27.4	1.0	19.2	2.3	26.6	1.0	17.9	2.0
8-9	27.9	1.1	20.2	2.3	27.2	1.0	18.6	2.1
9-10	28.4	1.1	20.9	2.4	27.8	1.0	19.4	2.1
10-11	28.8	1.1	21.7	2.5	28.2	1.0	20.1	2.2
11-12	29.4	1.1	22.4	2.5	28.7	1.0	20.8	2.3
1 year	29.7	1.1	23	3	29.3	1.0	21	3
1½	32.3	1.1	26	3	31.9	1.1	24	3
2	34.5	1.2	28	3	34.1	1.2	27	3
2½	36.3	1.3	30	3	36.0	1.4	29	3
3	37.8	1.3	32	3	37.5	1.4	31	4
3½	39.3	1.4	34	3	39.1	1.5	34	4
4	40.8	1.9	37	5	40.6	1.6	36	5
4½	42.1	1.9	39	5	41.7	1.7	38	5
5	43.7	2.0	42	5	43.8	1.7	41	5
5½	44.7	2.0	44	5	44.5	1.8	43	5
6	46.1	2.1	47	6	45.7	1.9	45	5
7	48.2	2.2	54	7	47.9	2.0	50	7
8	50.4	2.3	60	8	50.3	2.2	58	11
9	52.8	2.4	66	8	52.1	2.3	64	11
10	54.5	2.5	73	10	54.6	2.5	72	14
11	56.8	2.6	82	11	57.1	2.6	82	18
12	58.3	2.9	87	12	59.6	2.7	93	18
13	60.7	3.2	99	13	61.4	2.6	102	18
14	63.6	3.2	113	15	62.8	2.5	112	19
15	66.3	3.1	128	16	63.4	2.4	117	20
16	67.7	2.8	137	16	63.9	2.2	120	21
17	68.3	2.6	143	19	64.1	2.2	122	19
18	68.5	2.6	149	20	64.1	2.3	123	17
19	68.6	2.6	153	21	64.1	2.3	124	17
20-24	68.7	2.6	158	23	64.0	2.4	125	19
25-29	68.7	2.6	163	24	63.7	2.5	127	21
30-34	68.5	2.6	165	25	63.6	2.4	130	24
35-39	68.4	2.6	166	25	63.4	2.4	136	25
40-49	68.0	2.6	167	25	63.2	2.4	142	27
50-59	67.3	2.6	165	25	62.8	2.4	148	28
60-69	66.8	2.4	162	24	62.2	2.4	146	28
70-79	66.5	2.2	157	24	61.8	2.2	144	27
80-89	66.1	2.2	151	24	—	—	—	—

deviation of 2.6 inches. Seven sources, listed below, were used; at least 9 other large series of white Americans were discarded, including several groups of Air Force and Navy flyers, submariners, airline pilots, and university students—all too highly selected to represent Americans generally.

1) The least stature, 68.2 ± S. D. 2.6 inches, characterized the largest group, 97,795 selective service registrants of 1943-44 (Karpinos, 1958). This series was certainly representative, but measuring techniques left much to be desired. More importantly, the data are now 16 to 17 years old. During the 25 or so years between the two World Wars, the height of both selective service registrants (Karpinos, 1958) and soldiers (Randall, 1949) increased by 0.7 inches, while current evidence (recalculated from Damon, 1957; Newman, 1960) indicates a continuing trend. If this is assumed to be at the same rate, 17/25 of 0.7, or 0.5 inches, should be added to the 1943-44 figures, giving 68.7 inches.

2) The greatest stature, 69.5 ± S. D. 2.4 inches, was reported by Tyroler (1958) for two groups of paper industry workers, 316 from North Carolina and 377 from Texas, measured between 1952 and 1958. These men were largely Old Americans of British or Northwest European descent, taller by about 0.5 inches than Americans generally (Brues, 1946) and from regions of the country where—doubtless due to the predominance of such strains—white registrants (Karpinos, 1958) and soldiers (Brues, 1946; Newman and Munro, 1955) were taller than the general average. In fact, only the highly selected flyers and college students (Sturzebecker, 1950; Elbel, 1954) exceeded these paper workers, who are therefore decidedly taller than average Americans.

3) In the United States Army, average heights of 68.4 or 68.5 inches (S. D. not given) were reported by Randall (1947, 1949) for each year from 20 through 24. These 21,110 soldiers were 0.2-0.3 inches taller than draft registrants of corresponding age (Karpinos, 1958) not only in the 20-24 group, but at 18-19 and at 25-29 as well, due partly to selection for health and partly to minimum Army height limits of 60.0 and at other times 60.1 inches. Projecting for these subjects, measured in 1946, the assumed rate of stature increase: 14/25 of 0.7, or 0.4 inches, added to 68.5 gives 68.9 inches as the expected height for present-day soldiers. Subtracting 0.2 inches, as a correction factor for the greater height of soldiers, one again obtains 68.7 inches as a reasonable estimate for the height of white American males aged 20-24 years.

4) A smaller but more recent series of 693 recruits aged 20-25 years

entering a Naval Training Center in Illinois during 1952 averaged 68.9 ± S. D. 2.54 inches in height (Gibbons *et al.*, 1953). There was a lower limit on height, presumably 62.0 inches. Two-thirds of the men were Middle Westerners, taller than the national average (Newman and Munro, 1955; Karpinos, 1958); so here again, as an estimate for the general population, the figure of 68.7 inches seems reasonable.

5) In 1954, a group of 210 Army recruits, aged 20-24, at Fort Dix, New Jersey, averaged 69.0 ± S. D. 2.65 inches tall (computed from original data of Damon, 1957). Stature was measured against a wall, giving values some 0.4 inches above those obtained by measuring rods in the other surveys cited. Most of the recruits were from New York, New Jersey, and New England, in that order, and hence shorter than the national mean—by 0.4 inches (Brues, 1946); 0.2 to 0.3 inches (Newman and Munro, 1955); or 0.5 inches (Karpinos, 1958). If the technical and geographic factors are regarded as cancelling one another, so also may the 0.2 inches for long-term height increase and for superiority of Army recruits to civilian population. This leaves a high mean of 69.0 for the group—evidence for a continuing stature increase in the military population.

6) A group of 86 professional bus and truck drivers measured in 1950 and 1951 averaged 68.9 ± S. D. 2.4 inches tall (computed from the original data of Damon and McFarland, 1955). Included among them were 20 Texans, averaging 69.1 inches; the rest, including 44 men from New England and 22 from every other region of the country, were 68.8 inches tall. Adding an increment for stature increase since 1951 would bring them up to or over 69.0 inches. Although heavy vehicle drivers comprise the largest distinct occupational group in the country, the extent to which they represent the general population in height is uncertain.

7) The final comparative series is Pett and Ogilvie's (1957) excellent Canadian survey, with a mean value of 67.9 inches (no S. D. given) for Canadian men aged 20-24, a weighted figure derived from 68.5 inches for 132 Canadians exclusive of Quebec, and 66.6 inches for 115 men from Quebec, predominantly of French descent. We consider Canadians outside Quebec comparable to the United States population, and indeed at several age groupings they fell only 0.1 to 0.2 inches short of the American stature. Once again, therefore, 68.7 inches (68.5 + 0.2 inches) seems a reasonable figure for the United States.

So much for mean values. The standard deviation of 2.6 inches is that of the selective service, or general population (Karpinos, 1958), with the other series quite close.

After each tabular entry was thus tentatively established, it was then examined in relation to the preceding and following entries. Trends within each of the separate studies were used as a guide, even though no one or combination of them quite represented the present American population. Only in age and sex categories where data were sparse did the final "smoothed" entry differ by more than 0.2-0.3 inches or 2-3 pounds from the preliminary figures.

Our method was unavoidably subjective, within the indicated limits, though the correction factors mentioned as well as others at different ages or for females were consistently applied. We believe that deviations from "true" values attributable to the method rather than to shortcomings in the data will be well within the tolerance range for most practical applications of height and weight data. To repeat, table 1 is not intended as definitive. It is offered as a reasonable synthesis, for workers who need it, of data heretofore scattered or unpublished.

NOTES

A few points should be kept in mind in evaluating the tabular data. They are intended as national averages, from which specific groups may well diverge. The number of subjects at the various ages ranged from a few million men of military age, and several hundred thousand school children, to a few hundred in the seventh and eighth decades. Data for 70- and 80-year-old men were derived largely from a single study (Damon and Stoudt, 1959) eliminating subjects with marked kyphosis or pitting edema. The questionnaire survey of Master, Lasser, and Beckman (1960) furnishes "provisional" height and weight averages for several thousand persons between the ages of 65 and 95, but in view of the highly selected sample and the uncontrolled measuring technique of thousands of respondents, certain entries in the 9th and 10th decades are best left blank for the present.

Age: Children from birth to six years of age were usually measured within a few days of achieving the indicated age, whereas age thereafter generally referred to last birthday.

Race: Although the subjects are described as white, some Negroes were included in a few of the samples used for the younger age groups. At these ages, however, Negro and white children of comparable socioeconomic status do not differ in height or weight (Meredith, 1959).

Techniques for measuring height: Below age 6, recumbent length is customarily used, giving mean values some 0.5 inches greater than erect stature in boys and about 0.75 inches greater in girls (Vickers and Stuart, 1943). When a person stands against a wall straining for maximum height, he is taller than when measured standing erect but free. In men, both old and young, the difference averaged 0.4 inches (Damon and Stoudt, 1959; Kennedy, 1960). The wall measurement is preferable.

Clothing: Table 1 presents nude values. Only those studies were utilized in which subjects were measured nude or where valid correction factors could be applied.

The use of the standard deviation as a measure of dispersion for weight should be regarded with caution because of the skewed nature of the distribution. Percentiles are better, but are infrequently available. For stature the distribution is more normal, and the standard deviation can be safely used to indicate dispersion.

SUMMARY

Based on both published and unpublished data, "average" heights and weights are presented for white males and females in the United States from birth to old age.

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AGE CHANGE IN OBESITY[1]

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LIFE insurance company statistics suggest the lethal effect of overweight (Dublin and Marks, 1952; Dublin, 1953; Evans, 1947). For males the death rate doubles with a 50% increase in weight, although for females the effect of overweight is less severe. There is thus a direct relation between increasing weight and increasing mortality and further correlation with body build and age: overweight is particularly lethal to tall young males and perhaps also to short women over 30 (Dublin and Marks, 1952). The data suggest many mechanisms for this effect of excess weight, including raised blood pressure with atherosclerosis (*cf.* Murrill, 1955; Barr, 1953; Keys, 1955) linked with fatal clot or hemorrhage in myocardium or brain, or with chronic nephritis, or breakdown of pancreas or liver in sugar and fat metabolism. Excessive fat increases surgical risk in gall-bladder and appendix operations. The cancer death rate is not raised by overweight and that from pneumonia, despite the apparent reduction in vital capacity, is raised less than the accidental death rate. Many overweight or fat people reach old age though obesity and longevity clearly do not go together (Pearl and Moffett, 1939; Swanson *et al.*, 1955). Since arterial disease is usually the actual killer (Keys, 1955), the correlated overweight may have little or no causal role. Susceptibilities to coronary disease or to atherosclerosis plausibly are inherited aspects of a metabolism which also tends

[1] For beginning the original Jefferson study of obesity as a collaborative effort as well as for their help in many phases of it I should like to thank Dr. Karl Paschkis, Dr. Robert Matthews, Dr. Paul Swenson, and Dr. Abraham Cantarow, respectively heads of the Endocrine Clinic, and Departments of Psychiatry, Radiology, and Biochemistry. I am grateful to my technical assistants and especially to Mrs. Tamara Litwin, Mrs. Rosemarie Schroy, Mrs. Irene Gould, Miss Alice Winett, and Mrs. Dorothy Elicker who from 1952-1958 engineered the time-consuming routine of persuading subjects to return for re-study and carried out the studies on blood, photography and the statistics. Support for this study, as part of a long-term project on the anthropology of chronic disease, came from N. I. H. Grant A-224 and continuations. Though I have benefited from the advice of many people, including those mentioned, responsibility for opinions and conclusions is entirely my own.

toward overweight, but in which excess fat *per se* may be no more handicapping than excess lean weight (Keys, 1955).

Many people have assumed that the supposed effects of excess fatty tissue from overeating are reversible through loss of weight. Barr (1953) quotes Wilens on the possibility of reversing atherosclerosis in individuals just as it is reversed statistically in populations when a dietary reduction is forced on them (Dedichen *et al.*, 1951). Diabetes and hypertension and many other diseases are helped by weight reduction. The clearest statistical proof of possible reversibility of damaging effects of excessive fat is in the data of Dublin and Marks (1952; *cf.* Dublin, 1953) that mortality in a sample of "obese" who lost weight is only about 10% above normal in contrast to the 50-60% excess of the overweight as a whole. But selection for ability to lose weight may also select for longevity.

I ask the following questions: (1) Are those obese overweight people who die young distinctive enough so that one can predict their danger? (2) Are those obese who survive still handicapped after their rapid weight gain levels off and (3) do they age normally? (4) Are the obese, as a whole, constitutionally different from healthy people or is the 20% or more excess of fat their only distinction? Other problems, such as the unity of the syndrome of obesity in man and its genetic, physiologic, and social causes, remain for later analysis.

SAMPLES

To answer these four questions we studied in cooperation with the Endocrine Clinic and Departments of Psychiatry and Radiology at Jefferson Medical College from 1944-1948 a group of 124 "healthy" obese white adult females. A re-study of this group from 1954-1957 was supported by N. I. H. Grant A-224 and continuations. Forty-six returned for study, five were dead, and 73 were lost (including a group of 20 reached but refusing to return). We were not able to collect a matched control sample of healthy middle-aged women. We have only a small female sample for physiological comparisons. Hence for metric comparisons we fall back on series published by Bayer and Gray (1934) from the San Francisco environs, by O'Brien and Shelton (1941) from the country as a whole for clothing sizes ($N = 10{,}042$), more selected samples of young WAFs (Churchill and Bernhardi, 1957) and Smith College students (Steggerda, 1929), as well as whatever medical norms are avialable of middle-class American women. Control samples for age

change in whites are much scarcer. In addition to some data in the clothing survey of O'Brien and Shelton (1941) we can rely on the careful longitudinal data of Büchi (1950) on Swiss villagers though for fewer measurements than ours, and on the much more extensive but male cross-sectional study on Irish by Hooton and Dupertuis (1952).

ANALYSES OF PROBLEMS

Subjects dying between studies

As seen in tables 3 and 6 the sample of five dead (they died at an average age of 43) is too small for predictions: there are no siginficant differences from either the lost or returnee groups using the 1% level of confidence. The dead's body weight of 250 pounds exceeds the rest by more than one standard error, a deviation which could occur by chance about one time in eight. The higher blood pressure, high blood sugar, large circumferences, relatively longer and deeper trunk, broader head and face, etc. suggest a larger body bulk, implying cardiovascular overload, and retention of infantile body build to a greater degree than in the rest. If confirmed in a larger sample these trends would simply carry the observed distinctions of the obese in general to a greater extreme.

Handicaps of surviving obese

Table 1 lists those diseases or conditions which afflicted our sample at mean ages of 40 and 49 respectively (1944-48 compared with 1953-57). We do not have proper control data, since these are usually given as annual incidence in a population. For example the interesting analyses of the health of part of the Baltimore white population by Collins, Phillips, and Oliver (1951) allow only guesses about the total incidence of any given disease by age 45 since any chronic condition or even a disease which may repeat (*e.g.* pneumonia) or last several years will increase the estimate. For example Bayer and Gray (1934) in their sample of 100 healthy women of middle age record 18 appendectomies and 12 gynecological operations while the Baltimore sample by age 45 shows an appendicitis incidence of 78.3% and a gynecological disease incidence of 200% including here repeated sickness and surgery. Other data, for example those from surveys on prevalence of chronic and disabling disease quoted by Ciocco and Lawrence (1951) and Downes (1953) show that 75-80% of the population are free from chronic disease at age 45, so that at least 70% must be considered healthy. Such a conclusion

TABLE 1

Incidence of certain diseases or symptoms in obese white women as determined by current clinic diagnoses and by patients' histories (i. e. stated remembrance).

DISEASE OR CONDITION	RETURNEES 1944-48 INCIDENCE	RETURNEES 1953-57 CHANGE	EXPECTED CHANGE CONTROLS 40-50	LOST 1944-48 INCIDENCE	CONTROLS AGE 45 INCIDENCE
Cardiac	17%	+11%	+16%*	23%	10%*
Renal (nephritis, etc.)	4	+ 7	+ 6*	8	6
Hypertension	9	+30	+ 6	13	7
Pneumonia	24	+ 2	+ 5	9?	39-
Scarlet fever	20	0	0	5?	9
Appendectomy	15	+ 2	+ 2	15	18 [1]
Gall-bladder disease	17	+13	+22*	13	49—*
Diabetes	2†	+11	+ 1	3†	1+
All gynecological disease	63	+ 7	?	35?	12—200+*
Varicose veins	22	+19	+ 6	23	7
Arthritis	30	+31	+16*	37	17*
"Anxiety"	44	+ 6	+ 5*	32	19*
"Dizziness," "headache"	37	+17	+20*	31	37*
Dyspnea	9	+ 6	—	23	?
"Healthy"	17	—11	?	12	75

The data from the 46 returnees and 78 "lost" obese subjects certainly cover some inaccuracies, almost always omissions: probably all frequencies should be higher. Patients with developed diabetes or cardiovascular disease were excluded from the sample originally and the rise in diabetes and hypertension is striking. Control frequencies (columns 3 and 5) are derived by adding the yearly incidences from data of Collins, Phillips, and Oliver (1951), for a Baltimore sample studied for *yearly incidence* of disease: there is no way of properly controlling the repeated inclusion of the same individual with chronic disease.

* Hence the incidences marked with an asterisk should be divided by the average length in years of each disease, probably by 5.

[1] Frequencies from Bayer and Gray (1934).

† Incipient.

is more useful in relation to *individual* health, the ultimate concern in preventive medicine, than a statement in terms of annual incidence.

Our obese themselves are a selected sample, since subjects with major organic disease *at the time of study* were rejected, reducing the incidence of diabetes and cardiovascular disease. As many as 10-20% of our sample were "healthy" when first studied as contrasted with 6% of the returnees in 1953-57. This original level is low and decline rapid as compared with Public Health Service survey data. It is noteworthy that Bayer and Gray (1934) found no overweight person who could be included in their group of healthy working women. Clearly the general incidence of handicapping disease is much greater in overweight people, corresponding to the 58% deficit of "healthy" obese people shown in last line of table 1 (75% minus 17%). Though there is predominantly disease of the cardiovascular system, kidneys, liver, and pancreas the precise incidence of morbidity does not parallel the mortality excesses for obese females given by Dublin and Marks (1952), which stress heart disease, cerebral hemorrhage, nephritis, diabetes, liver and gallbladder diseases, and appendicitis and intestinal obstruction. These causes of death are, however, direct results of the kind of chronic pathologic handicaps under which our sample of obese suffers. Downes (1953) in the large Baltimore sample notes this association between overweight and an approximate doubling of incidence of the same chronic diseases (heart, hypertension, gall bladder, diabetes, arthritis), and Swanson and collaborators (1955) find a somewhat smaller increase, though rising markedly with age, in their Iowa samples. Yet this morbidity and its associated mortality are selective, not affecting all obese people and definitely affecting some overweight with little excess fat: association between overweight and subnormal bodily functioning does not prove a simple cause-effect relationship since clearly other (genetic?) weaknesses and strengths are involved too.

Worth noting in our sample, for example, is the incidence of scarlet fever in childhood as well as pneumonia in youth, paralleled by an extra high incidence of various gynecological conditions (from prolapses to uterine tumors and ovarian cysts) in adulthood. Obviously obesity places a mechanical strain on all blood vessels, including those of the heart, kidneys, and leg surface (varicose veins), inviting an increase in blood pressure with slower pulse for adequate oxygen supply. Mechanical stress on joints, hastening arthritis, and physiological strain on viscera with increased handling of fatty acids and glucose (*cf.* Hausberger and

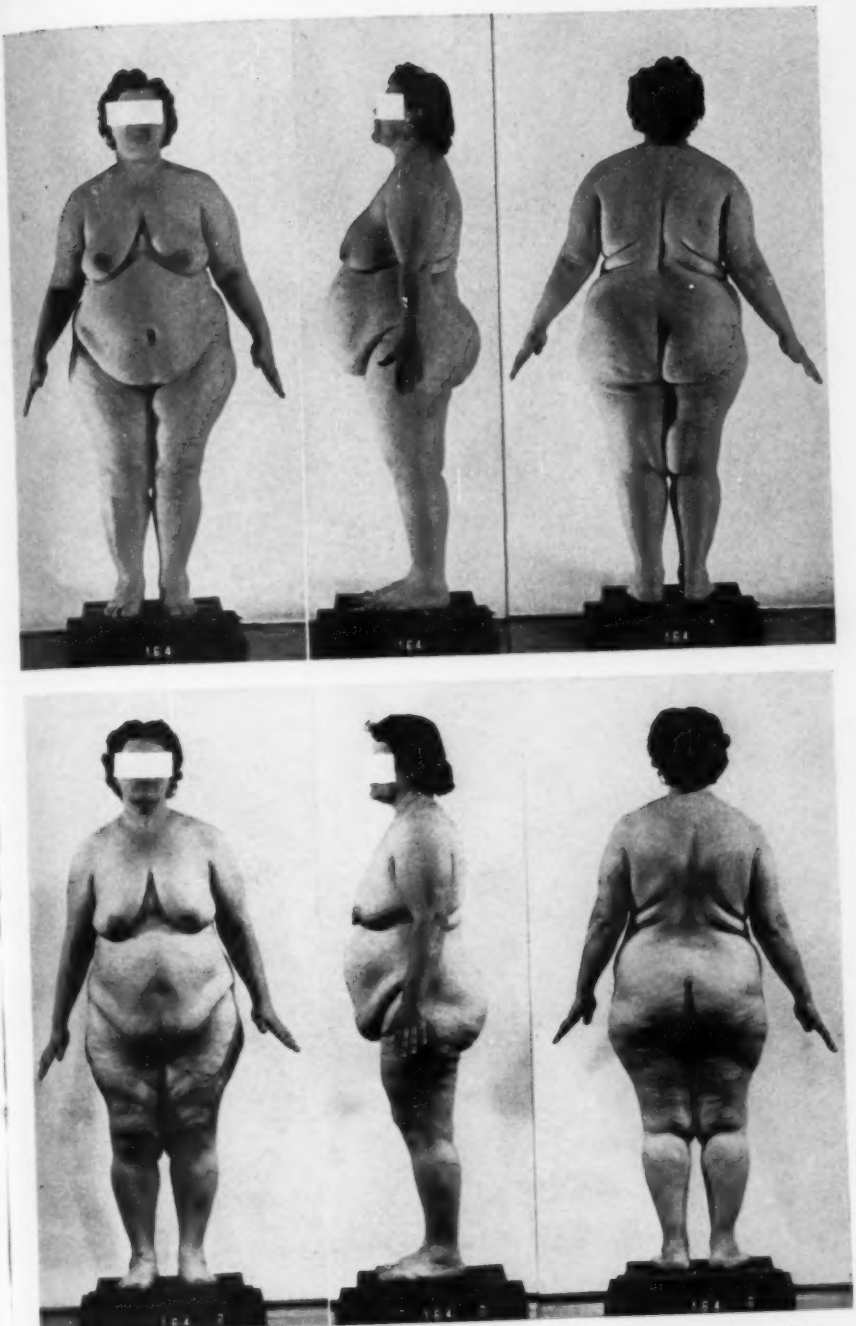

PLATE 1. #164 TYPIFIES OBESE AGE CHANGE, PHOTOGRAPHED AT AGES 42 AND 49 WITH 39-POUND WEIGHT LOSS DOWN TO 191 POUNDS (TWO YEARS LATER HAD REGAINED ALMOST ALL WEIGHT). SHE IS NEWLY DIABETIC, ARTHRITIC, AND VARIOCOSE.

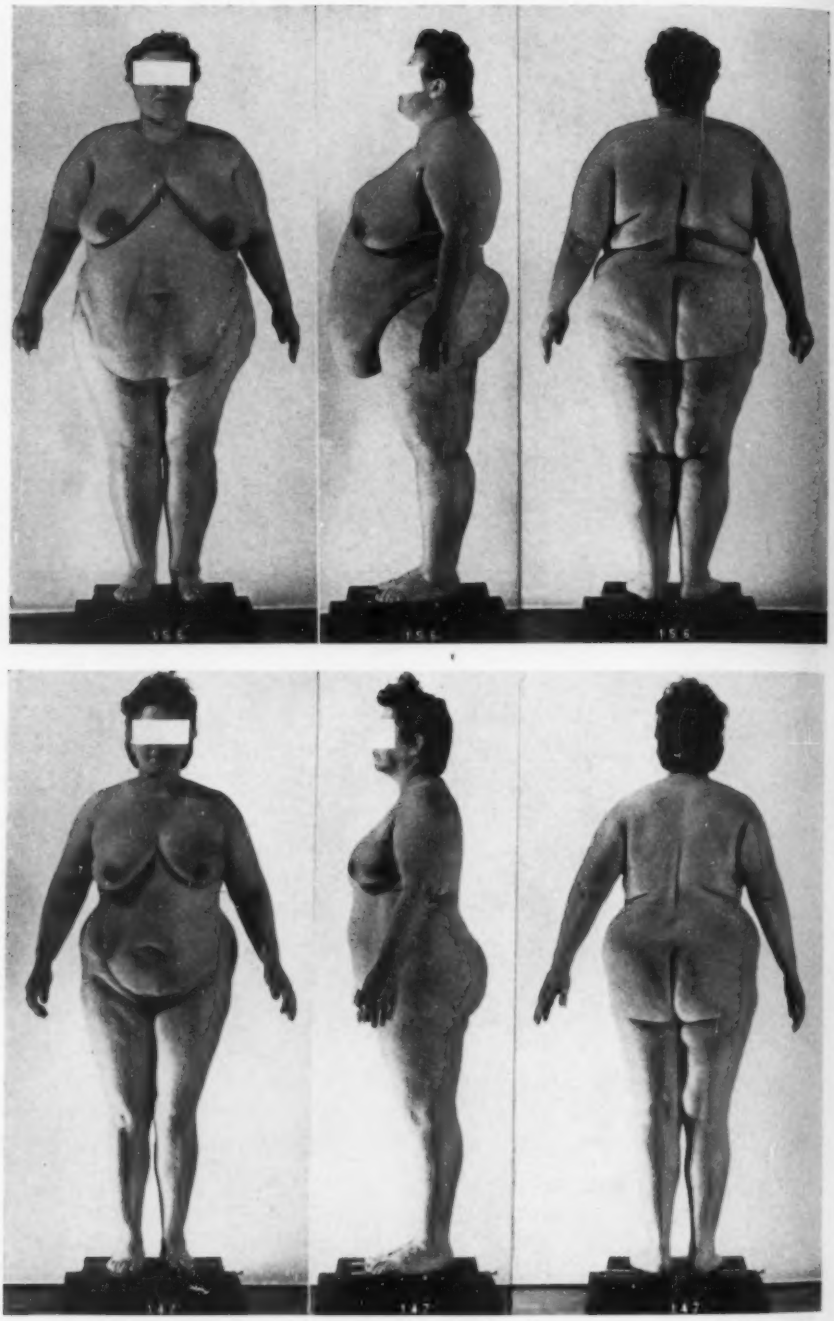

PLATE 2. #156, A 41-YEAR OLD INCIPIENT DIABETIC WEIGHING 315 POUNDS, DIED AT AGE 46.

#147, A 45-YEAR OLD WIDOW, TRULY OVERWEIGHT FOR ONLY TEN YEARS, COULD NOT BE REACHED FOR RE-STUDY.

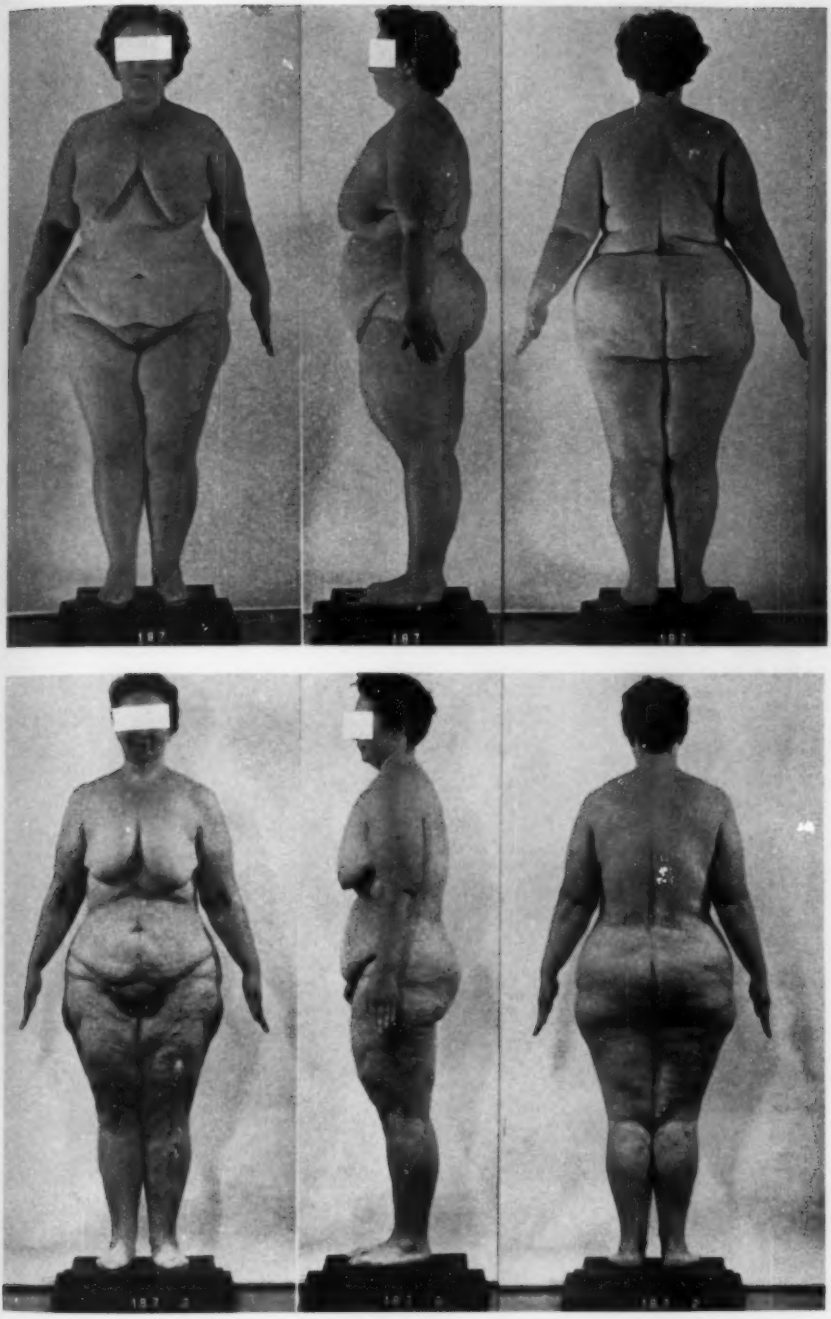

PLATE 3. #187, ONE OF THE FEW CHILDLESS OBESE, PHOTOGRAPHED AT AGES 35 AND 44, MAINTAINED A WEIGHT LOSS OF OVER 70 POUNDS (212 IN SECOND PICTURE) THROUGH PERSISTENT MEDICAL HELP.

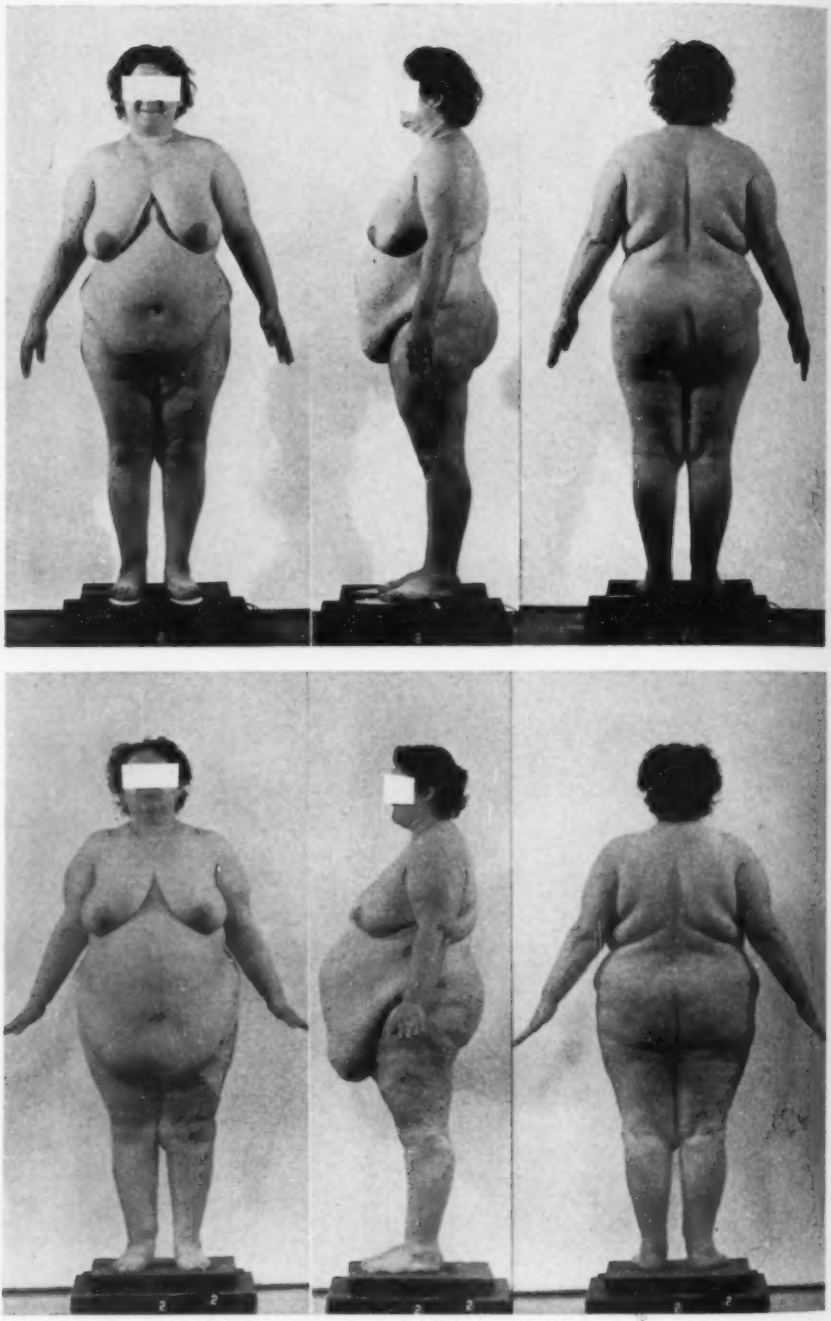

PLATE 4. #2, PHOTOGRAPHED AT AGES 32 AND 42 (206 AND 238 POUNDS), PERSISTENTLY GAINED WEIGHT AND BECAME DIABETIC, VARICOSE, AND HYPERTENSIVE WITH DAMAGE TO HEART.

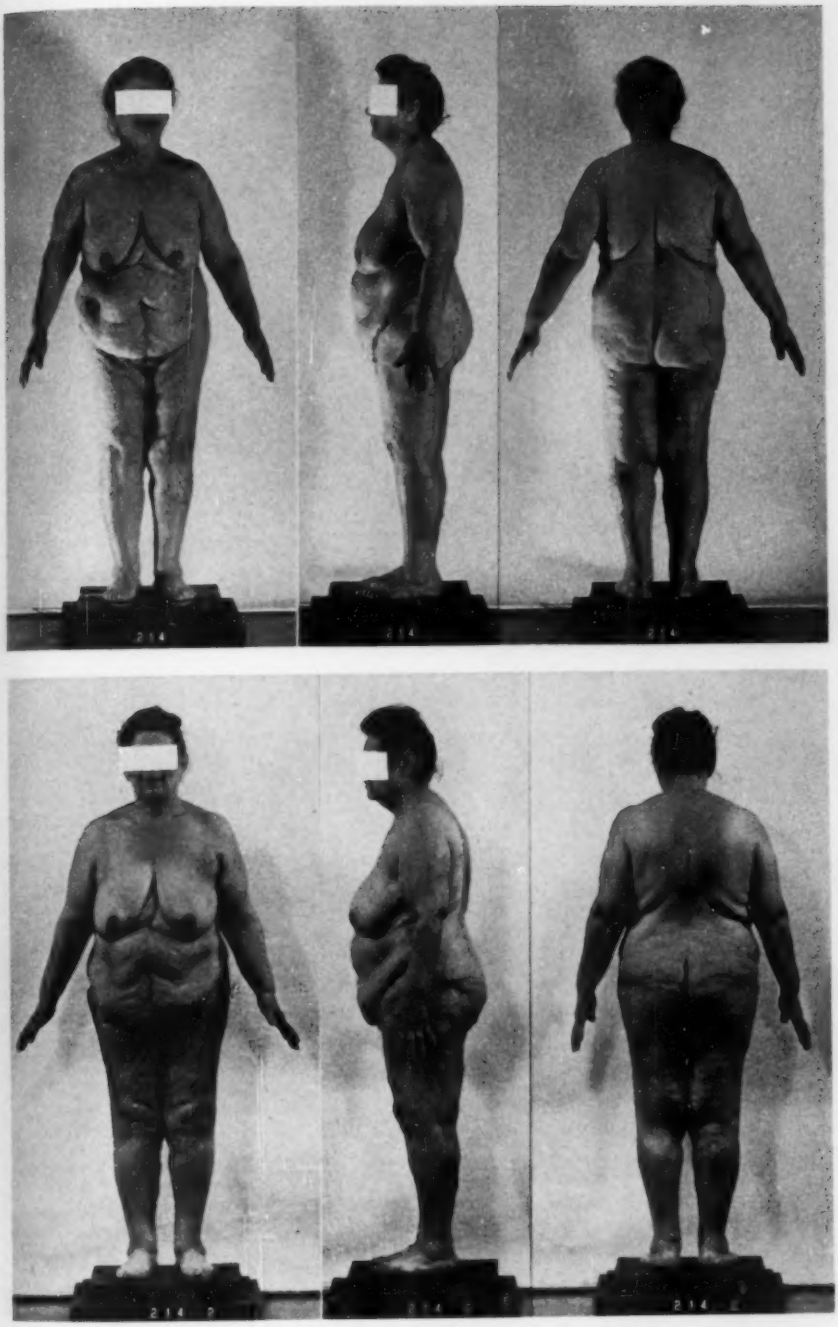

PLATE 5. #214, PHOTOGRAPHED AT AGES 60 AND 68 AT 171 AND 172 POUNDS, REMAINED ARTHRITIC AND VARICOSE AND BECAME HYPERTENSIVE: STATIC OBESITY ENTERING OLD AGE.

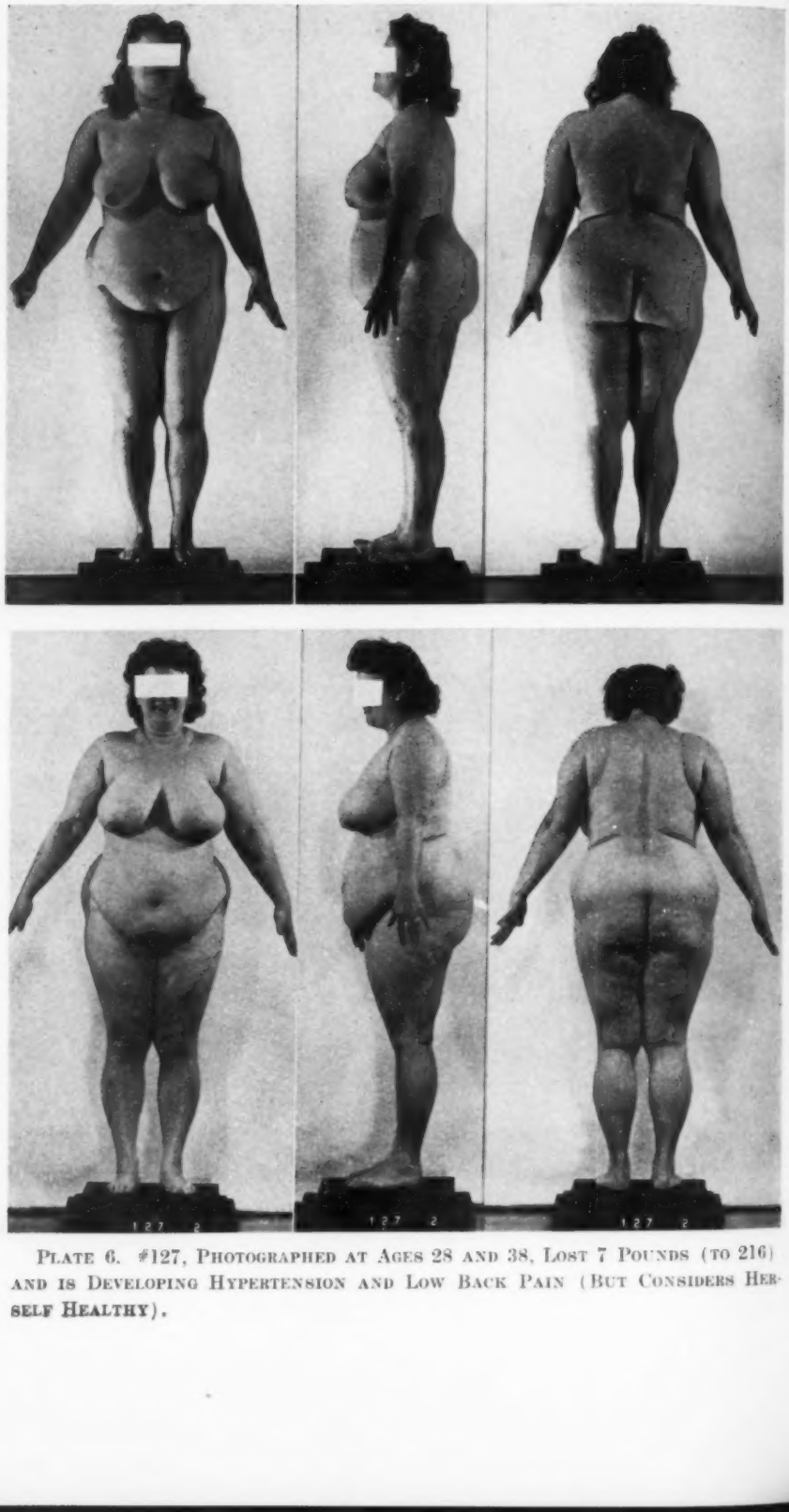

PLATE 6. #127, PHOTOGRAPHED AT AGES 28 AND 38, LOST 7 POUNDS (TO 216) AND IS DEVELOPING HYPERTENSION AND LOW BACK PAIN (BUT CONSIDERS HERSELF HEALTHY).

Hausberger, 1958) are likewise obvious. Dyspnea is a direct sequel of excessive fat around viscera. Dizziness plus headache, and nervousness and anxiety are less direct consequences of obesity, not ignoring the key observation that anxiety also plays a causal role (Bruch, 1950; Richardson, 1946; Schopbach and Angel, 1953). The data of table 1 indicate that at mean age of 39.5 each person had 3.1 complaints, of which 2.5 were chronic (compared with 2.5 and 1.9 for older controls): this list of handicaps is impressive.

Pattern and rate of aging

Even more impressive (table 1) is the marked increase in all these diseases seen during a 9-year period among the returnee sample. The increase is 1.5 complaints per returnee, slightly more than in an Iowa group 20% or more overweight (Swanson, *et al.*, 1955). Disease frequencies often more than double, and except for heart disease the increase is always greater than that expected on the basis of the Baltimore frequencies (Collins, Phillips, and Oliver, 1951). This is particularly striking for hypertension, varicose veins, arthritis, diabetes, and gall bladder disease. This accelerated susceptibility to disease is all the more emphatic in that the sample as a whole lost weight significantly (even though only six pounds) instead of gaining five pounds as the general population does. Simple maintenance of an extra 60-180 pounds of fat may be sufficiently extra strain to produce this extra morbidity, unless some of the obese inherit susceptibilities to age rapidly in certain physiological functions. An added strain is the variablility of weight of most individual obese. Thus the subjects report an average weight loss between studies of 46.4 pounds, with a range from 88 to 10 pounds. But there is an almost compensating gain, so that most subjects fluctuate in weight over a range of about forty pounds, certainly a metabolic strain.

Twelve subjects have never lost as much as 25% of their "normal" weight (average 22 pounds maximum loss) and in general have maintained or gained weight. Ten subjects with a maximum loss of 53 pounds have all held much of this loss, being 30 pounds lighter than when first studied (not necessarily at their maximum weight). But 24 subjects, with a maximum loss of 56 pounds, include 8 who have gained weight between visits; these 24 are the fluctuating group. We find no real differences between these three subgroups in body build, muscular strength, serum cholesterol level, or disease incidence. In all only 16 subjects gained weight between visits, 2 stayed constant, and 28 lost weight.

External factors at work in this include the social ban on obesity and the availability of psychiatric and other treatment for overweight.

Does selection by such external factors in any way bias our sample? Since the group of 46 returnees is the key in our study of age change we must compare the returnees with the lost subgroup. Table 2 shows an excess of 24% of widows, divorcees, and spinsters in the lost as compared to the returnee group. The returnees do not diverge significantly in social level, but do differ in being more successfully married than the lost sample. Changes of address linked with less success in

TABLE 2

Marital status and occupation of husbands of obese white women

STATUS	RETURNEES (46) 1944-48	RETURNEES (46) 1953-57	LOST (78) 1944-48
Unmarried (= no husband)	6.5%	4.4%	12.8%
Divorced or separated	4.4	4.4	11.5
Widowed	2.2	8.7	12.8
Married to:			
Executive	0	0	0
Professional or salesman	10.9	13.0	3.8
Service trades	13.0	15.2	14.1
Skilled worker	17.4	19.6	16.7
Maintenance, helper, etc.	32.6	26.1	18.0
Unskilled worker	4.4	4.4	7.7
Unemployed	8.7	2.2	2.6

marriage play a role in this distinction. Probably the lost are more recalcitrant and resistant to treatment although four of ten questioned by telephone claimed to have lost 40 pounds or more. Tables 3-5 show that in some physiological tests the returnees do not differ significantly from the lost. Metrically the returnees tend to be slightly smaller than the lost though this is statistically significant only for bi-iliac breadth and chin height.

To measure the existence of age changes we have used in tables 3-5 three times the standard error of the distribution of individual differences in each character on the null hypothesis that no change is expected. This assumption, reasonable for adults, would not apply to growth studies, however, where the individual differences would form a strongly skewed distribution. The individual differences include on the one hand the age change in tissue formation and posture of each subject and on the

other hand the personal variability of technique of the observer and random change in subject position. Technical and random changes cancel out. But they make the variance and standard error relatively larger than the "true" values for age change alone. If we estimate these technique-caused fluctuations in the age differences by comparing the standard deviation of age differences with standard deviations of the respective measurements (see table 4), we find the physiological measurements much more subject to random fluctuations than the morphological ones, as well as considerable contrast between such stable measurements as stature and variable ones as chest circumference, bi-iliac breadth, and face heights.

Many of the bony frame age changes are as expected (Hooton and Dupertuis, 1952; Büchi, 1950): stature and span both decrease, as disks and joints and muscles relax; chest depth (antero-posterior) increases, giving a relatively deeper chest and paralleling an increase in depth of abdomen. Head length rises and head height declines, and nose breadth and tip size increase. Though no standards are available one would expect the length of long bones to be stable through middle age; forearm length stays the same, but (upper) arm length decreases (most plausibly because of change in fat over the acromion process, its upper landmark).

Other changes do not agree with findings on normal people. One change is the decrease in chest breadth and chest circumference where a foreshadowing of the old-age increase in chest size should show up with a higher diaphragm and increased abdominal visceral mass. Probably two factors operate here: the absolute sag produced by the heavy fat on belly, breasts, and over the general waist area, and general slight decrease in subcutaneous fat thickness and turgor. This last is closely linked with the weight decrease; normally weight loss starts at about age of 60 (*cf.* Brožek, 1952; Swanson *et al.*, 1955). Also reflecting the weight drop are decreases in all circumferences and in bitrochanteric (but not bi-iliac) breadth and a slight shortening and increase in arch height of the foot. Likewise this slight loss in subcutaneous fat produces a drop in forehead and face breadths which normal series do not show until after 60. Broadening of the biacromial shoulder breadth needs explaining: apparently with aging the scapulae were held lower on the conical upper rib cage and hence farther apart, reflecting more relaxed trapezius and levator scapulae. In agreement with this is an 8 mm sag of the gluteal crease relative to the more solidly fixed popliteal crease; this indicates some loss of gluteus maximus tone. We might expect a

TABLE 3

CHARACTER	OBESE WHITE WOMEN: DEAD 1940s MEAN	RETURNEES 1944-1948 MEAN	1944-1948 S.E.	1953-1957 DEV.	1953-1957 S.E.	LOST 1940s MEAN	LOST S.E.	CORRECTED 1940s MEAN	HEALTHY CONTROLS MEAN
Age when studied	36.7	39.7	1.33	+9.35		39.7	1.13	*39.7*	36.2
Weight in pounds	242.8	218.1	5.33	*—5.85*	2.22	224.1	4.33	128.7ca.	135.4
Stature in cm	157.9	157.7	.84	*— .42*	.12	158.2	.76	*158.0*	161.5
Span	162.7	161.4	.82	*— .79*	.14	161.7	.91	*161.4*	164.0 [1]
Sitting height	87.2	85.5	.47	*— .37*	.18	85.8	.42	84.7	85.5
Bi-acromial breadth	37.5	36.9	.23	*+ .53*	.09	37.0	.19	*34.7*	35.5
Bi-iliac breadth	32.7	31.8	.25	*— .07*	.16	*32.6*	.23	*27.8*	30.1
Bi-trochanteric br.	37.1	36.0	.31	*— .69*	.15	36.3	.25	*32.5*	35.0 [2]
Chest breadth	29.8	29.6	.27	*— .74*	.17	29.4	.25	*26.7*	27.6
Chest depth (a.-p.)	24.5	23.6	.33	*+ .40*	.15	23.5	.26	*21.3*	20.6
Abdomen depth	29.4	28.1	.57	*+ .78*	.27	28.4	.46	23.8	
Arm length	29.8	29.8	.23	*— .34*	.11	29.8	.17	*28.7*	29.2 [1]
Forearm length (radius)	23.7	23.2	.14	+ .02	.04	23.2	.15	23.2	22.3 [1]
Foot length (sitting)	24.2	23.9	.16	*— .13*	.03	24.1	.14	*23.3*	24.0 [2]
Foot breadth "	9.3	9.3	.09	— .03	.03	9.3	.02	*8.6*	9.1 [2]
Arch height (standing)	6.4	6.7	.13	+ .03	.03	6.8	.10	*6.7*	7.1 [2]
Chest circumference	112.1	107.2	1.25	—1.28	.67	107.1	1.01	*88.2*	85.9
Waist circumference	109.7	103.4	1.58	*—2.34*	.77	103.8	1.46	*80.8*	73.2
Head circumference, mm	559.0	561.1	1.97	*—3.24*	.61	562.7	1.81	555.8	550.2 [1]
Head length	185.2	185.8	.77	+ .74	.14	186.6	.75	*184.3*	187.8
Head breadth (max.)	151.2	150.5	.72	— .06	.14	150.2	.56	148.3	148.8
Head height (auric-ver)	126.0	126.8	.74	*—1.02*	.41	127.7	.70	*126.4*	120.1
Minimum frontal br.	110.2	106.6	.62	*—1.41*	.35	107.5	.48	105.2	102.7 [1]
Face breadth (bizyg.)	140.8	138.3	.65	*— .60*	.17	138.0	.56	*135.1*	133.0

Jowl breadth (bigonial)	104.6	104.3	.68	*—1.48*	.41	104.7	65	101.5	100.4 [1]
Face height (nas.-men.)	115.0	114.5*	1.01	*—1.56*	.35	115.2*	.81	113.5*	114.1
Upper face height	66.4	69.0*	.64	*—1.54*	.39	69.1*	.50	*69.0**	71.4 [1]
Chin height	39.6	38.4*	.37	*— .44*	.20	*39.6**	.37	37.7*	
Nose height	49.6	51.0	.46	*— .96*	.24	50.7	.44	*50.8*	52.2
Nose breadth	33.0	33.1	.38	*+ .48*	.13	33.7	.35	33.5	33.5
Stature/$\sqrt[3]{\text{Weight}}$	10.0	10.4	.09	+ .02		10.3	.07	12.4ca.	12.4
Relative sitting ht.	55.2	54.2	.23	— .06		54.2	.18	*53.6*	53.0
Thoracic index	82.2	79.6	.84	*—3.58*	.49	79.9	.64	*79.8*	74.6
Cephalic index	81.7	81.0	.50	*— .40*	.13	80.6	.44	80.5	79.2
Facial index	80.9	82.2*	.70	— .15		83.8*	.70	84.0*	85.9
Nasal index	66.8	65.1	.86	*+2.23*	.38	66.7	.35	66.1	64.6
Number of subjects	5	46		46		78		124	100 var.

Note for Table 3: the base column for comparison in this table is the second one, showing mean measurements and indices of the returning sample of obese white females as studied between 1944 and 1948. Statistically significant deviations of the same sample re-studied 9 years later and of the non-returning, "lost" group given in column 4 are underlined. The dead, given in column 1, are also included among the lost. For the whole series of obese (N = 124) column 5 gives the "corrected" means after making allowance for extra fat, with significant differences from normal American women italicized. This control series is basically the sample of 100 *healthy* working women studied in San Francisco by Bayer and Gray (1934) with addition of (1) measurements of Smith College students by Steggerda, Crane, and Steele (1929), (2) measurements of equally young WAF trainees by Churchill and Bernhardi (1957), and (3) the huge clothing survey sample of American women by O'Brien and Shelton (1941).

* The number of measurements for face height (nasion-menton), upper face height (nasion-prosthion), and chin height for returnees are respectively 25, 26, and 32; for the lost they are 59, 60, and 68; for the total sample of the 1940s, corrected, they are 84, 86, and 100. Subjects with incomplete occlusion are omitted.

TABLE 4

Standard deviations to illustrate variability

CHARACTER	OBESE WHITE WOMEN: RETURNEES σ OF MEAN 1944-48	RETURNEES σ OF DIFF. 1953-57	LOST σ OF MEAN 1944-48	HEALTHY CONTROLS σ OF MEAN
Size of sample	46	46	78	100
Age when studied	9.03		10.01	10.34
Weight in pounds	36.14	15.02	38.27	15.31
Stature in centimeters	5.69	.84	6.73	6.18
Span	5.54	.98	8.00	6.70[1]
Sitting height	3.20	1.19	3.73	2.92
Biacromial shoulder br.	1.55	.62	1.66	1.37
Bi-iliac hip breadth	1.69	1.12	2.06	1.61
Bi-trochanteric hip br.	2.09	1.10	2.19	2.41[2]
Chest breadth	1.84	1.17	2.18	1.89
Chest depth	2.26	1.03	2.25	1.84
Abdomen depth	3.86	1.86	4.09	
Arm length	1.54	.75	1.47	1.68[1]
Forearm length (radius)	.95	.25	1.32	1.33[1]
Foot length (sitting)	1.08	.22	1.27	1.17[2]
Foot breadth	.60	.23	.64	.51[2]
Arch height (malleolus)	.87	.53	.87	.71[2]
Chest circumference	8.50	4.55	8.93	5.43
Waist circumference	10.73	5.22	12.86	7.14
Head circumference in mm	13.33	4.14	15.97	12.98[1]
Head length	5.22	.98	6.64	5.89
Head breadth	4.90	.94	4.92	4.30
Head height (auric.-vertex)	5.00	2.77	6.16	4.30
Minimum frontal breadth	4.21	2.40	4.27	4.94[1?]
Face breadth (bizygomatic)	4.41	1.16	4.99	4.24
Jowl breadth (bigonial)	4.64	2.80	5.78	4.39[1]
Face height (nasion-menton)	5.06	1.73	6.23	6.17
Upper face ht. (Nas.-pros.)	3.28	1.98	3.90	3.89[1]
Chin height	2.08	1.12	3.01	
Nose height	3.10	1.64	3.85	3.10
Nose breadth	2.56	.88	3.12	2.41
Stature/$\sqrt[3]{\text{Weight}}$	.61		.61	
Relative sitting height	1.56		1.63	1.38
Thoracic index (br. depth)	5.67	3.32	5.70	5.21
Cephalic index (lgth-br.)	3.37	.88	3.92	5.21
Facial index	4.08		5.38	3.78
Nasal index	5.82	2.57	7.54	5.88
Resting pulse rate	8.22	8.14	14.09	
Blood pressure, systolic	25.94	19.75	31.50	15.46
Blood pressure, diastolic	16.57	11.68	17.60	10.30
Blood sugar	19.26	24.86	24.43	
Serum cholestrol	50.65	55.23	58.68	39.52
Age at menarche	1.52		2.05	1.48

Note for Table 4: these standard deviations of the means of the second, third, fourth, and sixth columns of table 3 show the returnees to be less variable than the lost subgroup and the healthy controls to be less variable than the obese, especially in those measurements affected by subcutaneous fat thickness. The

similar sag in level of nipple measured from axillary crease. But this does not occur, probably because of shrinkage of fat in the breasts in many instances opposing the sag of the pectoralis major muscle, thus raising the variability of nipple position compared to gluteal crease sag with age. Standard deviations of individual differences are 8.4 and 6.5 mm respectively for these photographic measurements of gluteal crease and breast level.

If muscle loses tone and fibrous tissue loses elasticity prematurely in the obese, one might expect to find further evidence of acceleration of aging. Decrease in body weight might partly be such a process. But since there is no significant difference in age between those who lost and those who gained weight during the decade between studies it is equally probable that the mean weight loss is the end result of medical treatment which helped temporarily to reduce the weight of 80% of the returnees.

Reduction in sitting height normally should occur five to ten years later than in the obese, with the start of senile bulging, loss of elasticity, and vertical shrinkage of the intervertebral disks and loss of tone of the erector spinae muscle group (Hooton and Dupertuis, 1951, and unpublished observations on cadavera). Apparently sitting height decreases and vertebral and other arthritis increases in our sample because the fibrous tissue of the obese is inadequate for the excessive weight loads on joints. The increase in varicose veins and cardiovascular breakdown similarly suggests a premature loss of elastic strength of fibrous tissue.

This mechanism does not apply to the shortening of all vertical face and nose heights, a decline which is not normal until after the age of 60, together with the relative broadening of the nose. These changes in face form depend partly on loss of teeth and change in occlusion (though of course we excluded individuals who lost enough teeth noticeably to change their occlusion). The average of five teeth lost may be excessive for the 40 to 49 year age change, but comparison is difficult. Table 5 suggests that the obese in general have healthy teeth.

In morphology there is the normally occurring slight growth of ears

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obese are less variable than controls only in cephalic index and in long bone lengths. The second column gives the standard deviations of the distributions of differences between the same returnees measured nine years apart. This column shows that all physiological measurements, hip, chest, forehead and jowl breadths, and arch and upper face heights are changeable when re-measured; on the other hand stature, sitting height, span, shoulder breadth, forearm length, foot and head lengths, and breadths of head, face and nose are relatively constant when re-measured.

and nasal tip. Table 5 shows a slight increase in skin vascularity, as observed also by Hooton and Dupertuis (1952), and a peculiar shift in iris color. A chi-square test showed no relation between increased

TABLE 5

Observations on pigmentation, teeth, and blood groups and taste-blindness of obese white women

CHARACTER AND DEGREE	RETURNEES 1944-48	RETURNEES 1953-57	LOST 1944-48	CONTROLS*
Skin vascularity				
Slight	4.4%	0	6.5%	
Medium	41.2	34.8%	48.0	
Above medium	4.4	19.6	7.8	
Marked	50.0	45.6	37.7	
Iris color				
Brown	10.9%	15.2%	11.5%	11.0%
Light brown	4.4	2.2	7.7	6.8
Gray-brown	15.2	4.4	7.7	4.6
Blue-brown	17.4	4.4	12.8	27.1
Green-brown	30.4	47.8	26.9	17.8
Gray	0	0	2.6	4.0
Blue	13.0	17.4	19.2	15.5
Green	8.7	8.7	11.5	13.2
Iris yellow pigment				
None	30.4%	19.5%	41.0%	71.9%
Trace	23.9	17.4	10.3	
Slight	28.3	19.6	21.8	
Medium	17.4	37.0	21.8	28.1
Marked	0	6.5	5.1	
Dentition, individually				
No. teeth unerupted	.6	.6	.3	
No. teeth lost	13.7	18.8	12.0	
No. teeth carious	5.9	3.9	5.3	
% teeth with lesions	63%	72%	54%	
All upper teeth lost	24%	37%	21%	43%
All lower teeth lost too	9%	24%	10%	21%
Blood group				New York
O		36.4%	(40)%	42.7%
A		27.3	(20)	38.0
B		25.0	(40)	14.2
AB		11.3	0	5.1
Number tested		44	10	4,370
P T C Tasters	67.7%	73.9%	60.0%	70.0%
Number tested	31	46	65	—

* Control data on iris pigmentation come from a recombining of the categories of Brues (1946); those on teeth are from Morris' Human Anatomy, 11th ed., p. 1322 quoting Brekhus (1929) for clinic patients; the PTC tasting frequency is that of Snyder (1951), p. 499, and blood group frequencies of New York with Brooklyn are done by Wiener (1945).

vascularity as judged visually and gain of weight. Sixteen individuals were classified differently as to iris color on the second visit. In seven cases the shift involved changed judgment of brown pigment (3 to blue and two to brown previously classed as mixed, and two into green-brown from blue and green previously); since lighting was better the second time these probably represent correction of errors. In the remaining nine cases the change is recorded as an increase in yellow pigment (eight added to green-brown and one to green). What connection does this have with the rise in level of serum cholesterol shown in table 6? Another possible association with cholesterol and yellow pigment is development of arcus senilis at the corneo-scleral junction in eight subjects (17%), suggesting again an acceleration of aging. But within our returnee sample there are no significant associations between yellow iris pigment, level or degree of change in serum cholesterol, arcus senilis, and ABO blood type.

Premature aging in physiological function is equally definite (table 6). The rise in blood pressure, averaging 5-6 mm of mercury for systolic and diastolic pressures, is an example of this expected from life insurance company data on obesity. In normal people with loss of weight no rise in blood pressure with age is expected (Murrill, 1955). In pulse rate the decline of six impulses per minute when resting (sitting in a chair after being measured) probably exceeds normal change at this age: plausibly a mechanical effect of increased area of capillaries and raised blood pressure.

Rise in level of blood sugar is a classic change in chronic obesity, even though glucose tolerance tests on the group when first studied were "within normal limits." The observed rise of less than 20 mg% is an underestimate, since several of the mild diabetics in the series were being treated with insulin.

Serum cholesterol level behaves similarly. The standards of Keys (1950) and especially those Adlersberg *et al.* (1956) show a particularly noticeable rise in serum cholesterol during the period of middle age, especially in females (the male rise is earlier and males do not show the old age increase in serum cholesterol typical of females). Adlersberg *et al.* publish an increase of 25 mg% (224.5 at age 38-42 to 249.5 at age 48-52), while our sample by age 49 shows an increase of over 60 mg%.

The overproduction of insulin in some types of experimental obesity and its fat-synthesizing role (*cf.* Mayer, 1955; Hausberger, 1958) may suggest that pancreatic islets and liver tend to break down in human

TABLE 6

CHARACTER	OBESE WHITE WOMEN: DEAD 1940s MEAN	RETURNEES 1944-1948 MEAN	S.E.	RETURNEES 1953-1957 DEV.	S.E.	LOST 1940s MEAN	S.E.	COMBINED 1940s MEAN	HEALTHY CONTROL* SERIES MEAN
Age when studied	36.7	39.7	1.33	+9.35		39.7	1.13	39.7	40ca.
Age at menarche (stated)	14.3	13.1	.22			13.3	.26	*13.2*	13.6
Resting pulse rate	86.5	79.5	1.64	*—6.32*	1.63	80.3	2.01	80.0	78-80ca.
Systolic blood pressure	153.5	135.3	4.38	+5.17	3.34	140.1	4.10	*138.3*	125.4
Diastolic blood press.	100.0	87.1	2.80	*+5.83*	1.97	87.6	2.29	*87.4*	78.7
Blood glucose mg%	127.0	89.3	3.46	*+17.22*	4.46	90.6	3.10	*90.2*	86ca.
Serum cholesterol	216.0	205.8	9.10	*+65.6*	9.92	208.7	8.38	207.6	224.5
Total N	5	46		46		78		124	various

Significant deviations of the obese from controls or in age changes are italicized. Age is true chronologic age, that is as stated to the last birthday plus .5 years.

* Control ages for beginning of menstruation (menarche) come from Bayer and Gray's (1934) 100 healthy working women; blood pressures are interpolated to age 40 from Master, Dublin, and Marks (1950) and because of the prevalence of overweight in the American population are presumably a little too high for healthy norms, *cf.* Murrill (1955); serum cholesterol is from Adlersberg, *et al.* (1956) based on a Staten Island population and using the Sperry-Schoenheimer technique, whereas the present studies used the Lieberman-Burchard method with the Evelyn colorimeter.

The Number reported for Returnees and Lost, where less than the total, are as follows: for pulse rate 25 and 49 respectively; blood pressure 35 and 59; blood sugar 31 and 62, serum cholesterol 31 and 49.

obese only because they are overworked by increased food intake and increased sugar, fat, and steroid metabolism; but we have no direct evidence on the normal functional capacities of pancreas, liver, thyroid, or other exocrine and endocrine organs. Possibly in the obese many endocrine relationships, including the pituitary-gonad interaction, start relatively early and are subject to breakdown earlier than normal.

Menarche at 13.1 years shows the expected acceleration (*cf.* Bruch, 1939), but menopause (47.3 as stated by 18 subjects) is late (*cf.* a mean age of 43.5 in 26 women studied by Bayer and Gray, 1934); this parallels the observation of Hunt *et al.* (1958) that obese boys start adolescence early, but finish it slowly and irregularly.

Thus in general, age change in the obese is irregularly accelerated physiologically, morphologically, and pathologically.

Constitutional distinctiveness

We cannot conclude, just on the above evidence, that this acceleration is basic and not directly the result of the excess load of fat. But there are other constitutional distinctions (Angel, 1949) which do mark the obese when we remove the fat statistically (tables 3-5) and perhaps physiological distinctions too (tables 6 and 7). Table 5 shows an excess of yellow pigment and an excess of blood group B, a genetic distinction (Angel, 1957) not attributable to ethnic origins of the sample.

A comparison of X-ray measurements of bi-iliac breadth and chest breadth in four obese and four controls (somewhat younger) done with the help of the Radiology Department under Dr. Swenson in 1947 showed mean tissue thicknesses of 53.6 and 44.6 mm for the obese at the level of the iliac crest and the eighth rib as compared with 17.2 and 10.4 in the controls. Under caliper pressure these two thicknesses respectively decreased to 26.5 and 16.7 mm in the obese and 2.6 and 3.5 mm in the controls. In relation to the total measurement these thickness differences gave correction factors of 14% for hip breadth and 9.5% for chest breadth. Body weight is corrected by 42% ($3 \times 14\%$), sitting height by 3%, circumferences by $\pi \times 6$–7%. Head and face measurements are corrected for 1 mm extra fat thickness except in the lower face where 1.5 mm is assumed. The results are not perfect: obviously the effect of internal abdominal fat is not removed in waist circumference and abdomen depth measures, while correcting body weight by a tripling of the linear excess probably does subtract this internal excess fat too. It is worth noting that correcting the cube root of weight by 14% and

TABLE 7

Bone, muscle, and fat plus skin measurements and responses to certain stresses of obese returnees and a normal control group

CHARACTER	OBESE RETURNEES	NORMAL WOMEN
Age when studied	*49.1*	36.3
Stature in centimeters	*157.3*	163.3
Weight in pounds	*212.3*	132.6
Number studied	46	30
Biepicondylar breadth elbow	66.6 = 62.4 [2]	61.9
Biceps breadth, contracted	61.9 = 49.9 [1]	54.6
Biceps, contr.-relaxed difference	*23.0*	32.7
Right hand grip strength	25 kilograms	27.9
Thickness of fat + skin folds, Harpenden caliper, 10 g./mm²		
Arm, anterior midpoint	*15.5*	7.9
Arm, posterior midpoint	*27.7*	17.4
Back subscapular area	*35.9*	14.7
Abdomen, near iliac spine	*36.7*	18.7
Calf, lateral below knee	19.5	18.4
Climb of 10′ up staircase with 15 steps: effect on pulse rate		
Free climbing time, seconds	*11.6* seconds	8.0
Pulse rate standing	*89.8*	84.6
Pulse rate increase	*16.6*	24.0
Pulse increase, 11.5 sec. climb	16.6	16.3
Pulse rate resting in chair	72.8	74.9
Fingertip temperature (degree F)	88.1	90.8
Oral temperature (degree F)	98.3	98.2
Timing of vascudarity shift after scratch and blanching of forearm skin		
Reddening (flare: histaminergic?)	*6.2* seconds	7.7
Peripheral white (sympathetic?)	*25.3*	22.1

Significant differences between the obese returnees and the normal sample are italicized. The normal control sample, all from Philadelphia, are higher in socio-economic status than the obese (and include more women of East European origin and fewer of Italian and Irish origin than the returnees).

[1] Assuming that true thickness of a double layer of fat is 1.3 × the pinch-caliper value at 10 g./mm² (Garn, 1956) the thickness of a single fat + skin layer over the biceps brachii is 12 mm in the obese and 6 mm in the controls, so that a 12 mm reduction in biceps breadth of the obese is indicated for comparative purposes.

[2] Where marked caliper pressure is used, in the biepicondylar breadth measurement, the correction is taken as 35% of 12 mm or 4.2 mm.

cubing the result gives a weight of 141.2 pounds, which is probably about the lowest weight attainable by a reducing program for such women. Through these "corrections" we can approximate the body build of the obese without excess fat in order to compare their build with that of normal women in good health.

The corrected measurements show a relatively long trunk and short stature (and absolutely short upper and lower extremities), with narrow

trunk breadths but large chest and abdomen depths and hence relatively large circumferences and deep chest. Hands and feet are small and the arm short relative to forearm. The head, average in size, is short and rather high, with a wide-cheeked and round face, having a short nose and upper face. These shifts from the average American woman are all in the direction of childhood, as if the obese retained a somewhat more childlike body build than usual. The excess amount and trunk concentration of fat might even appear as a retention of an infantile condition. In terms of somatotype (Sheldon, 1941) the sample is overwhelmingly endomorphic. Somatotype averages 5.5-3.7-1.5 and shows exceedingly weak influence of ectomorphic tendencies (only in midline face or distal extremities) but almost average development of the mesomorphic component (especially the muscular part of this). This simply repeats in other terms what we have just pointed out as a retention of childlike or infantile body proportions and tissue masses.

For the returnee sample we can take up the proportions of tissues somewhat more objectively. Table 7 shows an elbow breadth which when corrected for overlying fat is average, confirming the impression from radiographs (*cf.* Reynolds, 1946), from somatotype photographs, and from subjective inspection that the obese have average rather than heavy bones. The contrast between relaxed and contracted biceps and the contracted breadth are less than the control group values taken on women of more elongated build. Yet hand strength of the obese is not significantly below normal. On the other hand the subcutaneous fat thicknesses measured with a pinch-caliper of 10 gm/mm² force (*cf.* Brožek, 1956) are double those of the control group. The striking fat increases are in the trunk area and back: the increases follow a definite pattern (*cf.* Garn, 1955) with the leg showing the least change and an exaggeration of the normal female age-fattening (Škerlj, Brožek, and Hunt, 1953). Here too, the obese show accelerating aging.

There remains the estimation of the amount of internal fat. Škerlj, Brožek, and Hunt (1953) give subcutaneous fat pinch measurements (taken with a caliper exerting a force of 5-7.4 gm/mm²) larger than our control group (table 7) by about a third, in accord with the difference in caliper pressure. These authors estimate that the *total* fat of their sample amounts to 21.9% of the body weight of 135 lbs., or about 30 lbs. Assuming a similar fat weight in our control sample this points to a mean weight of 105 pounds for muscle, bone, and viscera in our controls and somewhat less in our obese sample, in accord with their shorter stature. Hence the obese sample's fat weight must be 110-120 pounds,

or almost four times normal. Since the subcutaneous fat thickness is hardly twice that of our control group the visceral fat excess of the obese must be proportionately greater; this is suggested also by the trunk circumferences, by X-rays which show relatively high placing of stomach despite the sagging belly wall, and by subjective observation in the dissecting room. This visceral fat, tightly packed around pericardium, kidneys, adrenals, and pancreas, as well as in the whole mesentery should interfere with the mechanical and vascular efficiency of these structures and put stress on the quick venous shift of blood to or from the vertebral venous plexuses (Batson, 1940 and 1960) which compensates changes in chest and belly pressures.

The actual insulating effect of fat plus the raised mass-surface ratio in the overweight should mean a more efficient than normal retention of heat (*cf.* Baker and Daniels, 1956). Yet the contrast between core and fingertip temperatures is not significantly greater in the obese than in controls (*cf.* Adams and Covino, 1958), even though the controls were studied in May to August when room temperature at 75-85°F was 0-5° warmer than in winter. The obese show (table 7) relatively quick reddening ("flare") of the skin and relatively slow spread of surrounding white area after a skin scratch. This temporal separation of the last two phases of the Lewis triple response test indicates extra responsiveness of parasympathetic as opposed to sympathetic nervous system which might favor less efficient than usual adjustability to temperature change. Possibly sympathetic underactivity explains the diminished response of the obese pulse rate in stair-climbing: the obese must work harder than controls to walk or stand with 80 extra pounds and this is reflected by a standing pulse averaging five beats faster than that of controls; yet after doing 800-foot-pounds more work than controls in stair-climbing the obese show an average increase in pulse rate no greater than that of controls. But since table 7 shows a standing pulse rate 17 beats above resting pulse while the controls' standing-resting contrast is only ten beats it is possible that the heart in the obese is nearer its mechanical upper limit and cannot accelerate proportionately to increased work load for this reason.

The obese pulse response, therefore, may not reflect autonomic balance. But there are two subjective observations pointing to autonomic imbalance (parasympathetic dominance?). One is the relative immobility and economy of movement of the obese, with suppression even of the normal fidgeting motions during conversation. The other is the raised appetite level following morning anorexia.

In summary, there are many indications that the obese are biologically distinct from non-obese in growth pattern and perhaps in autonomic balance in the direction of the major human evolutionary change: towards pedomorphism.

SPECULATION

In this study of obesity we have three central observations, none of them new: the obese show increased sickness and death rates, increased speed of aging, and retention of characters of infancy and childhood. They also show raised variability (table 4), partly explained by the skewing effect of fat on measurement distributions.

This lack of homogeneity in the group selected as too fat means that one could predict only uncertainly for example, from early puberty and relative shortness of limbs relative to trunk, that an apparently average youth would become obese in adulthood. Variation in time of onset likewise is a factor associated with morphological variability, since those fat since birth (33%) are more endomorphic (Angel, 1949). This suggests that different expressions of a fundamental shift in growth rate, an acceleration of aging throughout life, could explain the variability as well as the morphological distinctiveness.

This shift in growth rate is not a simple speeding up. Early maturers are in adolescence relatively bigger, heavier, more muscular and fatter than normal (Reynolds, 1946) and are both shorter-legged and heavier as adults (Tanner, 1955, p. 72 ff.). But there is no strong correlation between early maturity and degree of adult fatness; and conversely, adolescent endomorphs, unlike those of mainly muscular build, are not true early maturers: they start adolescence early but complete it variably and late (Hunt, 1956). This observation suggests a mechanism for retention of certain infantile traits without the longevity increase which accompanies this evolutionary trend in man. What appears to have happened in human, as opposed to general mammalian growth, is (1) an increase in speed of growth especially in early life, (2) a slowing down in postnatal tissue differentiation, and (3) a *delay* in maturation which partly counterbalances this slowing. What appears to happen in obesity is a further increase in the first two of these but a partial return to the early maturation and death, the faster life cycle, of non-human mammals: something nearer the typical mechanism of infantilisation. If the pituitary-hypothalamic blood humoral circuit is involved here (an increased feedback sensitivity to other endocrine secretions?) then that

which is basic (and inherited?) may be simply an increased mass and sensitivity of the anterior hypothalamus, in turn affecting (a) growth, (b) anabolic autonomic balance and (c) satiety in different individual degrees and times depending on other genetic and environmental influences.

There remains the question of extra disease and death in obesity and their connection with more rapid aging. Life insurance data allow the inference that overweight is the direct causal factor and that removal of excess fat and fluid will slow aging. "We are eating ourselves to death" (Dublin and Spiegelman, 1952). This is a partial explanation. If it were a complete explanation, obesity would affect people almost alike, accompany no differences in build, and its removal would reduce excess mortality to normal. Some fraction of the obese person's typically greater sensitivity to disease might arise, then, as an area of lessened resistance involving decreased maximum effectiveness of liver, pancreas, kidneys because of too rapid growth as opposed to embryonic differentiation.

This discussion raises three questions. (1) What genetic mechanisms underlie obesity? (2) Is extreme obesity a disease of lipid metabolism, on the gene-enzyme equivalence hypothesis in which some as yet unidentified enzyme is inhibited (or lacking) in viscera and brain (*cf.* Snyder, 1959), with effects on hypothalamus and satiety secondary results? (3) Can we find any markers which will distinguish early those potentially obese who would be the first to die?

CONCLUSIONS

(1) In a sample of 124 obese white females studied in Philadelphia at the Jefferson Medical College, the rates of death and of chronic disease are raised.

(2) Study of 46 returnees a decade later shows an average speeding up of the aging process in morphology and in the underlying physiology, so far as measured, and with the reservation that the group is at least as variable as the general population.

(3) As a group the obese are distinctive in keeping many features of childhood in their basic body structures (in addition to their food-addiction, economy of movement, and probable excess of blood group B).

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BRAIN WEIGHT AND BEHAVIOR[1]

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IN the February, 1959 issue of *Human Biology*, Spuhler discusses the physical conditions that he believes are prerequisites for the full acquisition of that variety of culture realized by members of the genus Homo. These prerequisites are (1) accommodative vision (2) bipedal locomotion (3) manipulation (4) carnivorous-omnivorous diet (5) cortical control of sexual behavior (6) vocal communication and (7) expansion of the association areas in the cerebral cortex. After some discussion of the role of each of these factors in the evolution of man's capacity for culture, Spuhler makes a most interesting observation about the relation between man's acquisition of culture and his ontogenetic developmental pattern. Specifically, Spuhler says "Man, then, is not much different from other primates, especially the apes, in the general sequence of events from conception to birth. After birth, the ontogenetic pattern in man differs markedly from that of all non-human primates but differs in a direction forecast by the general trend of primate evolution. I would guess that this elongation of the life periods after birth is a consequence of physiological adaptation to the acquisition of culture. Culture is a biological adaptation with a non-genetic mode of inheritance depending on symbolic contact rather than fusion of gametes. It has greatly supplemented somatic evolution. In all known human societies, individuals participate in social systems whose members represent more than a single biological family in which all are connected (as the social insects are) by gametes from one parental set. No human family is a self-sufficient system of social action. Symbols rather than gametes make this so. It may be assumed that the genes controlling the growth cycle in man have been changed through selection to man's human, cultural environment."

If the idea that the acquisition of culture played a crucial role in the elongation of the human maturational periods after birth is correct, it may furnish a clue to the answer to the question of whether or not any particular fossil primate group was on the evolutionary line leading

[1] This problem was suggested by Dr. S. L. Washburn, Professor of Anthropology, University of California, Berkeley, California, and is a part of a program investigating the origin of human behavior supported by the Ford Foundation.

to man. If the primate was advanced in motor development and soon on it's own, the group probably was following the ape ontogenetic pattern. If, on the other hand, these infants were slow in development, it may be assumed that much longer maternal care was demanded, and that the group living patterns were evolving along the human pattern. If motor development is closely related to brain development, and it is the thesis of this paper that this is the case, it is possible to predict behavior from brain development. As soon as more infant primate skulls are found, it will be possible to estimate the brain capacity, and from this, behavior.

There is no direct way of reconstructing the behavior patterns of the fossil primates. Various inferences about the behavior of certain groups, such as the australopithecines, have been made from the physical characteristics inferred from skeletal remains (Bartholomew and Birdsell, 1953) and from bone and stone from australopithecine sites which may or may not be artifacts (Dart, 1960). These must forever remain reconstructions, since there are no australopithecines for us to observe. However, there is evidence at hand that primate behavior does develop at a relatively constant rate related to the percentage of adult brain weight achieved. For example, the infant chimpanzee and the infant human both acquire the ability to hold their heads erect when their brains have achieved a weight of between 40 and 45% of adult weight.

A more thorough discussion of the data that allow for the above statements is necessary at this point. The pertinent data were gathered for other purposes, but can be put to use in the present context. They are (1) the data on the development of the brain of the chimpanzee as summarized by Schultz (1940), (2) the data on the development of the brain in man from Denzer, Koningsberger, and Vonk (1941), (3) the data on the development of the brain of the macaque as determined by Zuckerman and Fisher (1938), and (4) the data on the age of appearance of ten behavioral traits in macaque, chimpanzee, and man as reported by Riesen and Kinder (1952).

These traits are (a) arms extended, prone, (b) raises head and upper chest, (c) rolls to prone, (d) head steadily erect (sitting), (e) rests on hands, (f) stands on fours, (g) walks on fours (creeps), (h) sits unsupported, (i) bipedal standing, (j) bipedal walking.

The table below shows these traits, by the letter preceding them above, as they appear in monkey, chimpanzee, and man in relation to the percentage of adult brain weight attained.

Perhaps the most interesting discrepancy in the appearance of these

traits is in trait c (rolls to prone), which appears in the chimpanzee at 46% of adult brain weight and does not appear in man until he has achieved 57% of adult brain weight.

Behavior and brain weight as a percent of adult brain weight

	a	b	c	d	e	f	g	h	i	j
Monkey	37	37	37	37	37	37	37	41	41	
Chimpanzee	41	41	46	41	46	51	51	53	62	64
Man	44	39	57	42	46	58	59	46	62	64

Ratio of monkey to man .73 Ratio of chimpanzee to man .96

Rank-order coefficient of correlation between chimpanzee and man .88

The extent to which these traits appear in the same chronological order can be measured by the use of the rank-order correlation coefficient. This index of relationship is .88, which is statistically significant. This means that the appearance of these traits in much the same order in chimpanzee and man is almost certainly attributable to something other than chance.

The average per cent of adult brain weight corresponding to the appearance of the ten traits (nine in the case of the macaque since trait j is not comparable) can be calculated by adding them up and dividing by ten or nine as the case may be. Ratios between monkey/man and champanzee/man can then be calculated by dividing the monkey and chimpanzee averages by the average for man. When this is done, the ratios are found to be .73 for monkey/man and .96 for chimpanzee/man.

Since this ratio and the correlation coefficient for chimpanzee/man is so large, it is possible to construct a generalized curve for the primates that reflects the three variables of age, behavior, and per cent of adult brain weight achieved. This curve is a sort of line of best fit between the human and chimpanzee developmental curves, and falls within the range of variability for both species. With this curve it is possible to predict with considerable confidence any one of the three variables if the other two are known.

The application of this generalized curve to ancient primate groups must await the discovery of fossil series that will give us some idea of brain weight at birth, at one or more points between birth and maturity, and at maturity. It is not necessary that such specimens be of any specific age, but only that they represent the age range from young to old.

Once such data is available, it will be possible by interpolation to calculate the percentage of adult brain weight which the particular form attained at various ages. With this information and with the generalized curve which may be derived from data already at hand, it will be possible to project the behavioral traits which the form was capable of performing at any state in its development. For example, if infant australopithecine skulls have relatively large capacities, approximating the 40% of adult brain weight characteristic of chimpanzees, and if they achieve adult brain weight by age four or five, it is probable that parental obligations were fulfilled early, and that *Australopithecus* was maturing along the ape pattern. This is probable because of the small capacity of adult australopithecine skulls and because of the small size of the brain of the original specimen from Taung. If, on the other hand, these skulls have approximately 25 to 30% of adult brain weight, and do not achieve adult brain weight until age seven or eight, *Australopithecus* was developing more slowly and more along the human ontogenetic pattern.

In addition to the further information needed on fossil forms, additional research is needed on the devolpment of the brain in the chimpanzee and the monkey. The size of the present sample is too small, and the groupings are much too gross. Furthermore, the behavior of the chimpanzee needs to be investigated from one year of age to maturity under the same kind of controlled, objective research design which Riesen and Kinder have demonstrated. With further research in these areas, and with the lucky find of a few more infant skulls, it will be possible to construct much more complete behavior curves to match brain curves, and thus to cast illumination upon the behavior patterns of ancient man.

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SEGMENTAL VOLUME REDUCTION BY LOCALIZED VERSUS GENERALIZED EXERCISE[1]

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A review of the literature on the value of generalized versus localized or so-called "spot" exercises indicates that this question has never been studied scientifically. In the opinion of Brobeck, Keyes, and Mayer (1956), "spot" reducing may be possible. These views were expressed in a symposium on weight reduction and were not supported by experimental evidence. Personal communication with several of the panelists confirmed the paucity of factual information on this subject. The purpose of this study was to investigate the relative value of generalized versus localized exercise in the reduction of weight and in changes in segmental volume.

PROCEDURE

The subjects for the investigation were undergraduate university women interested in weight reduction and willing to participate in the study which involved an exercise and diet regimen. Thirty-three subjects were selected from a group of 58 volunteers on the basis of height and weight. This was done for the sake of homogeneity of the sample. Subjects who were 5′ 10″ tall or shorter than 5′ 2″ were eliminated. In order to identify extremes with respect to weight, observed weight was compared with a standard by several different methods to determine relative weight. Those who were markedly (more than approximately 40%) or only minimally (less than approximately 10%) overweight were to be excluded from the study. Twenty-two subjects were then selected after a personal interview, on the bases of an evaluation of their apparent interest and cooperation, and the likelihood of their being able to follow the specified program throughout the entire experimental period. By the Shelton and Skeels (1951) standard of

[1] This work was supported in part by the Wisconsin Alumni Research Foundation and the National Institute of Arthritis and Metabolic Diseases, Grant A-2652.

optimal weight, these twenty-two subjects ranged from 7.72% to 40.71% overweight.

These subjects were then divided at random into the localized (Group I) and the generalized (Group II) exercise groups. A control group (Group III) of eight subjects was selected from volunteers who were overweight and desirous of reducing their weight. The control group received no systematic exercise and were not on the diet regimen. These subjects were interviewed and all agreed that to the best of their ability they would follow approximately the same daily diet and exercise program that they had followed in recent months. All participants in the entire study had received a health grade of "A" as certified by the University Division of Student Health.

An attempt was made to provide a controlled diet of neither negative nor positive caloric value. This was obviously only approximate for each individual. Recommendations regarding daily caloric intake for young adults in the age group of the subjects of this study range from approximately 2,000 to 2,400. There is some indication that the latter figure may be high. Therefore, a diet of from 2,000 to 2,200 calories was arbitrarily chosen as a reasonable approximation of probable energy needs. The diet was in part weighed and in part determined by volume measure, as estimated in the U. S. Department of Agriculture Handbook (1950). It averaged 2,070 calories daily and was followed for six weeks by the two experimental groups under the supervision of one or more of the investigators. Each meal was served in a special dining room of the University Student Union. At the end of each week, each subject signed a card indicating whether or not she had adhered entirely to the regulations in regard to diet, which involved eating all of the food served and no food or liquid of caloric value other than that in the specified diet. The deviations reported were minimal and considered to be of no definite importance.

The experiment continued for eight weeks. The first and eighth weeks were used for gathering the data; the exercise and diet programs were conducted during the second through the seventh weeks. Each of the two experimental groups had three 45-minute exercise periods weekly. Group I received localized exercise involving hip and abdominal muscle groups. Complete localization could not be obtained. The program for Group II included generalized exercises for the upper trunk, shoulder girdle, arms, and legs as well as those involving hip and abdominal muscle groups. The exercises selected were of the type usually administered for conditioning or reducing purposes. They can be performed in unison

and their speed and duration can be fairly well controlled. An experienced physical educator conducted the practice sessions for both groups. Both exercise programs demanded very severe physical exertion. Exercises for the two groups were matched in type in an attempt to equalize the severity of the work, and were planned to increase progressively in severity during the experimental period.

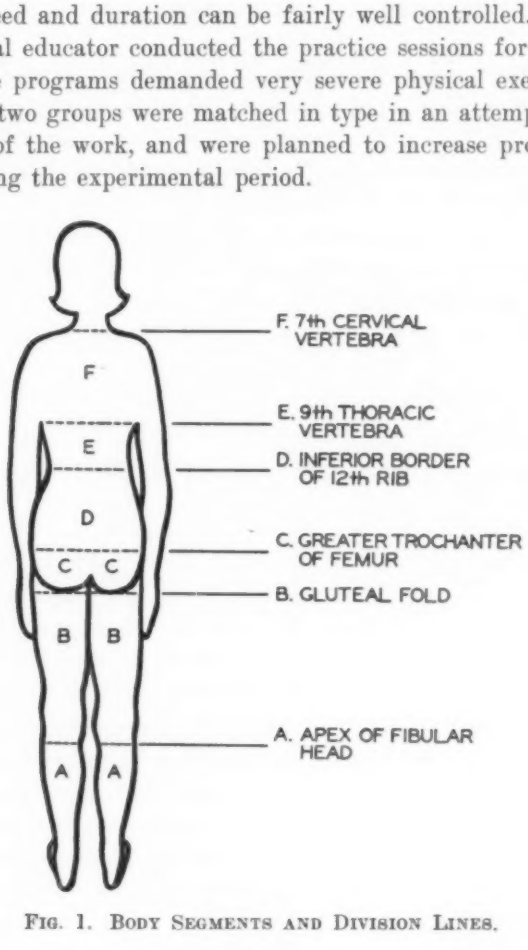

FIG. 1. BODY SEGMENTS AND DIVISION LINES.

Changes in body mass were measured by two methods. One was weight in kilograms. The second measure was volume determined by the amount of water displaced by various segments of the body. The technique of measuring segmental volumes was the same in principle as that used by Carns and Glassow (1957). Body volume was determined by measuring the amount of water displaced. Fractional volumes for the various segments were obtained by immersion to carefully localized landmarks. The accuracy of the method was increased by introducing a water gauge. This permitted the filling of the tank to predetermined levels and the accurate return to the original level as the

water displaced by the anatomical part under study was drawn off for weighing. The landmarks selected for segmental volume determinations were the apex of the fibular head, gluteal fold, greater trochanter of the femur, inferior border of the 12th rib, 9th thoracic vertebra, and the 7th cervical vertebra. (See figure 1.) The volume of the following parts could thus be measured: legs, thighs, gluteal region, hip region, lower thoracic region, and the combined shoulder and arm region. The landmarks were recorded in each case as the distance from the soles of the feet when assuming an easy stance. Insofar as possible the same posture was assumed inside the tank and checked by the same investigator. The procedure was modified for segment F in order to avoid submerging the head. In this case, with the subject in the tank, the water was brought to the level of the 7th cervical vertebra and drawn off to the level of the 9th thoracic vertebra. After the subject left the tank, it was again filled to the predetermined level of the 7th cervical vertebra and drawn off to the level of the 9th thoracic vertebra. By subtracting the first measurement from the second, the weight of the water displaced by segment F was established. Meticulous attention was given to the respiratory phase when measuring segments E and F. Observations were made at the end of the greatest expiration possible after a deep inspiration. These were repeated until a constant value was obtained. The water in the tank was maintained at approximately body temperature. The subject descended to a fixed platform at the base of the tank for each measurement. A harness which passed under the arms and was hooked over the side of the tank was used to allay any apprehension on the part of the subject.

Reliabilities were determined for the segmental volume measures in a preliminary study and training period. Reliabilities were estimated by analysis of variance techniques and ranged from .934 to .997.

RESULTS

The analysis of covariance was used to compare pre-exercise and post-exercise measures of segmental volumes and weight. Details of this technique are given in Cochran and Cox (1957). Table 1 gives the initial and final means for the segmental volumes of the three groups. It is evident from table 1 that the three groups differed somewhat on the initial measurements. The analysis of covariance permits an adjustment, employing the principle of linear regression of the final measures in terms of the initial measures and thus equates statistically the groups for initial status. The adjusted final means are given in table 2.

F-values were found to be significant beyond the 1% level for segment D ($F = 6.59$) and body weight ($F = 11.58$). All other F-values were not significant. The conclusion, then, is that the three groups differed significantly at the close of the study in body weight and in volume for

TABLE 1

Means of segmental weight and total weight in kilograms

	INITIAL			FINAL		
SEGMENT	GROUP I	GROUP II	GROUP III	GROUP I	GROUP II	GROUP III
A	6.91	7.01	6.47	6.81	6.91	6.46
B	14.54	14.19	12.08	13.87	13.72	11.88
C	8.30	8.12	7.08	7.98	8.06	7.30
D	12.20	11.19	11.42	11.34	10.44	11.42
E	7.42	6.83	5.87	6.96	6.45	5.84
F	16.18	16.35*	14.61	15.84	16.16*	14.82
Weight	70.00	68.45	62.33	67.27	66.60	62.37

* N = 10 (Complete data available for 10 subjects only).

TABLE 2

Adjusted final means

SEGMENT	GROUP I	GROUP II	GROUP III
A	6.73	6.74	6.80
B	13.17	13.31	13.41
C	7.63	7.87	8.05
D	10.81	10.83	11.60
E	6.40	6.41	6.66
F	15.55	15.74	15.74
Weight	64.77	65.59	67.19

segment D. As the table of adjusted means (table 2) indicates, the two experimental groups (Groups I and II) were similar in adjusted mean for segment D, but the control group (Group III), which had no special exercise and did not participate in the controlled diet, differed. For body weight, the two experimental groups differed somewhat, with the control group exceeding both of these. A test of the difference between adjusted body weight means for Groups I and II indicated that this difference was not significant.

This covariance analysis was done separately for each of the segments

and for body weight; this procedure ignores possible correlation between the measures of different segments. Another analysis, involving only the two experimental groups (Group I and Group II) was therefore made to test the hypothesis that these two groups differed in what is often called "profile"; technically this is the interaction of measures and groups. Greenhouse and Geisser (1959) gives procedures.

Since the experimental groups had been made up by random assignment, this analysis was made only on the final measures. Furthermore, the interest was primarily in the measures of volume of the six segments, and so body weight was not included. The results indicated no significant differences between groups, either in "elevation" or in "profile." Elevation corresponds to the sum of the volume measures. The finding of no significant difference in profile is of special importance, since this indicates that the effects of localized exercise as compared with generalized exercise were not manifest in segment-to-segment difference in the two groups. This finding is consistent with the similar adjusted means for Groups I and II given in table 2.

SUMMARY AND CONCLUSIONS

There is little evidence in the literature comparing the effects of localized or of "spot" exercise with generalized exercise. This study was designed to examine the effects of these two types of exercise on segmental volumes and body weight for college women. Thirty University of Wisconsin undergraduate women were the subjects. Twenty-two of these, who ranged from 7.72% to 40.71% overweight, were assigned at random to a localized (Group I) and a generalized (Group II) exercise group. These students followed for six weeks a diet regimen averaging 2,070 calories daily. During this period Group I received localized exercise involving hip and abdominal muscle groups; Group II received generalized exercises for the upper trunk, shoulder girdle, arms, and legs as well as those involving hip and abdominal muscle groups. The remaining eight students formed a control group which received no special exercise and did not participate in the diet regimen. Body weight and segmental volumes were measured for all 30 subjects before and after the six-weeks period. An analysis of covariance, holding constant initial measures, indicated that the control group differed significantly from the two experimental groups in body weight and in volume for segment D, the region from the inferior border of the 12th rib to the greater trochanter of the femur.

The post-exercise data for thetwo experimental groups were analyzed to determine whether or not there was a significant difference in the pattern or "profile" of segment volumes for these two groups. No significant difference was found. This finding is of special importance since this indicates that the effects of localized exercise as compared with generalized exercise were not manifest in segment-to-segment differences in the two groups.

This study, therefore, indicates first that exercise plus a diet regimen over a period of six weeks was accompanied by a reduction in body weight and in volume of segment D (the hip region) whereas this was not true for a control group which did not participate in either the exercise or the diet. Second, the analysis of segment volumes for the two experimetnal groups indicated no noticeable differential effect attributable to spot exercise.

ACKNOWLEDGMENT

The authors are indebted to J. S. Zahari, Food Unit Coordinator, University of Wisconsin Memorial Union, for his efficient cooperation in arranging very satisfactory facilities for serving special meals, and to P. J. Clearly, Chef, for his excellent planning of a well balanced diet, including very close approximation to the requested total daily caloric intake and unusually well designed variety in food.

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BOOK REVIEWS

Anthropologia Helvetica I. Die Anthropologie der Eidgenossenschaft (Anthropology of Switzerland as a whole): A. Text, 700 pp., 499 tables, 144 figs.; B. Atlas, 161 maps, 2 × 504 photographs. Art. Institut Orell Füssli, Zürich, 1956.

The monograph deals with the investigation on Swiss conscripts, carried out from 1927 to 1932. Further publications will be devoted to cantons and geographical regions ("Landschaften").*

Chapters I through IX describe the organization of these large-scale investigations, the training of the investigators, the research program, and the record blank. We are told how the subjects were photographed, how they were studied and how the material was processed. Prof. Schlaginhaufen was aided by a staff of 12 assistants, 159 field investigators, 4 photographers, and a secretary. Chapter X is devoted to the methods (mostly R. Martin's) and deals with the instruments and measurements as well as with descriptive characters. Chapters XI and XII give the recapitulation and report on the organization of the investigations in the respective locations.

Chapter XIII deals with the results; it makes up almost the whole book. The average age of the 35,511 investigated men was slightly over 19 years. The mean values of the 35 metric characters and indexes are shown in Table 489 (p. 689). The measurements and indexes are compared with the mean values of other populations.

Table 499 (p. 690) summarizes the frequency data for five descriptive characteristics: the profile of the nose is straight in 53%, convex in almost 20% and concave in 16% of the investigated subjects; the rest had other forms. The profile of the occiput is mostly (64%) rounded; it is straight (vertical) in 9% and very rounded in 9%. Wavy hair of various degrees appears in 16%, whereas 80% had smooth ("schlicht") hair. Eye color was brown in 27%, "meliert" (intermediate) in 47%, and "light" (blue, gray) in 25%. The hair was blond in 31%, brown and dark brown in 63%, red in 3%; the rest had other or mixed colors.

The author combines two, three, four, five, and six characteristics in many tables, without computing intercorrelations. On the basis of

* Anthropologia Helvetica: II. Die Anthropologie der Kantone und natürlichen Landschaften. A. Text, 709 pp., 970 tables. B. Atlas, 7 pp., 63 cartograms. Art. Institut Orell Füssli, Zürich, 1959.

the combinations of six characteristics (stature, cephalic index, morphological facial index, nasal index, eye and hair color), the author tries to provide a racial analysis of Swiss conscripts following Deniker. As he found only 1.57% of "Nordics (Homo Europaeus)," 0.28% "Ost-europide Rasse (Homo Vistulensis)," 0.61% "Ibero-insulare Rasse (Homo Mediterraneus)," 1.41% "Alpine Rasse (Homo Alpinus)," 2.47% "Litorale Rasse (Homo Atlanto-Mediterraneus)," and 2.31% "Dinarische Rasse (Homo Adriaticus)"—altogether 8.66% "pure" representatives—the results seem meager. Over 91% of the men were "mixtures" of the six hypothesized races. Again, it is only fair to say that the author recognizes that those "pure" racial representatives are "pure" only in the sense of the combinations of the six characteristics chosen. No genetic "purity" is anticipated. Nevertheless, on p. 691 he speaks of the 91% as "Kreuzungsprodukte der sechs Rassen"—an apparent oversimplification, to say the least.

The aim of Schlaginhaufen's monograph is, as he puts it, to present the mean values, variability and geographical distribution of the 40 characteristics, as well as a set of their combinations. This aim was achieved, though with more modern statistical methods the results could be presented in a shorter and more informative manner.

In the Atlas (B) 161 maps are devoted to the distribution of single traits (1-72) and to combinations of traits (73-161). An immense labor, indeed! Even more important, as an anthroposcopic source, are the photographs of 504 individuals (profile and face). These only serve to confirm the immense variability of the Swiss population—but, where in Europe (or at least in Central Europe) is there no such variability?

This brings us to a crucial point: do "races" exist at all among the Whites in Europe? Despite Deniker and many other physical anthropologists of the older generation, I doubt it. There is a white racial stock, there may be local types, selected (probably long ago) under the impact of natural environmental factors and (later, more and more) of social institutions (languages, class barriers, etc.) which impose certain restrictions to "free" intermingling, but which, on the other hand, are rarely if ever insurmountable. It appears that there simply are no (and never were) "pure races" among the European Whites.

Unintentionally, Schlaginhaufen's monograph is a proof of this. Nevertheless, the monograph is a valuable source book of "classic" anthropology in R. Martin's sense. Between the completion of field work and the publication of the results, more than 15 years have elapsed

and this review appears more than thirty years since the plans for the investigations were laid out, thirty fruitful years during which new questions and new views as well as new methods in physical anthropology and statistics were developed. Deniker's (though perhaps then the best) classification of "races" is obsolete today—as are many others, including my own developed during the thirties. Yet, this large and carefully written monograph will have lasting value as a standard for comparisons. It is an eloquent testimony to the indefatigable spirit of the Nestor of Swiss anthropologists, Professor Otto Schlaginhaufen.

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The Jaws and Teeth of Primates: Photographs and commentaries. By W. Warwick James. xii + 328 pp. Pitman Medical Publishing Co., London, 1960. £5. 5. 0.

For the many anthropologists who have no easy access to comprehensive collections of primate skulls, this new publication will be particularly useful. For every genus of recent primates a typical adult skull of at least one species has been selected from the vast material at the British Museum. There are nearly 250 excellently reproduced photographs. Of each specimen three views are given (including a scale): Side view of the entire skull, with teeth almost in occlusion, base view of skull, and top view of mandible (all focused on the dentition). The 64 species shown include even such rare forms as *Urogale* and *Callimico*. For many genera skulls of both sexes are represented. In addition, a number of very instructive views of primate dentitions (with bone removed to expose the roots *in situ*) are presented in large size, the preparation of an aye-aye being specially noteworthy. The accompanying text describes the characteristic features of the dentition and jaws and, more briefly, of the skull. It also discusses other bodily features, the distribution, habits, etc. of each genus, though in a somewhat random fashion.

Three brief appendices deal with the individual development of the jaws and teeth, the temporo-mandibular joint and (by Mr. L. G. Smith of the London Zoo) the diet, care and longevity of monkeys and apes.

From the bibliography of only four pages it is evident that this book does not claim to have dealt with primate odontology in an exhaustive manner. It is the result of the enthusiastic interest and devoted labor of its octogenarian author who has shared with us his extensive, careful observations, collected notes and a unique album of photographs of primate skulls. For quick reference these photographs are far more convenient than would be the skulls themselves and there are few places which can boast of such a complete collection. Considering the great number and of perfection of the illustrations the volume is reasonably priced and will be useful as a reliable aid in identifying primate material.

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The Antecedents of Man. An Introduction to the Evolution of the Primates. By W. E. LE GROS CLARK. vi + 374 pp., 152 figs. Edinburgh University Press, 1959. 21 s. Quadrangle Books, Chicago, 1960. $6.00.

Only four years since the publication of his text on the fossil evidence for human evolution, Professor Sir Wilfrid Le Gros Clark has brought forth a most admirable work on the comparative anatomy of primates. It is well written and up-to-date with regard to the inclusion of recent findings and the interpretation of their significance. This useful book is the culmination of substantial original work by the author and it brings together the body of knowledge regarding morphological relationships of living and fossil primates. This study of evolutionary morphology has "been adapted for use as an introductory textbook which is intended for students of whom some may have no special knowledge of comparative anatomy." While the book adequately fulfills this purpose by briefly describing the general mammalian characters of each anatomical system discussed, there is no lack of well presented detailed information of value to the student with a background in human anatomy.

Users of Le Gros Clark's first book, *The Early Forerunners of Man*, will realize that this is essentially a revision of that out-of-date and long out-of-print classic. Errors have been corrected, interpretations based on misconceptions and on dubious fossils have been omitted, and many

parts are wholly new, particularly with regard to the Hominoidea, including man. The basic outline of the 1934 work has been preserved, except that material on the tree-shrews is no longer isolated in a separate chapter but is included within the appropriate portions of the text. There are 63 additional illustrations and at least 30 others are redrawn and provided with more adequate captions.

After a completely new introduction on evolutionary process in which emphasis is placed on "total morphological pattern," the second chapter discusses the taxonomy of the Order *Primates,* following Simpson (1945). A discussion of the various anatomical systems of both living and fossil forms follows in seven separate chapters on the dentition, skull, limbs, brain, special senses, digestive system, and the reproductive system. A chapter on the evolutionary radiations of the primates concludes the book. This last section points to the various trends in primate evolution, aiming to apprehend the precise relationship of one group with another. The distinguishing characteristics of each group are discussed and then summarized. The change in emphasis since 1934 is particularly noteworthy on two points. The African Miocene remains and the Australopithecines have provided considerable new evidence for interpretations, especially among the higher primates, and the point of view is of a more functional nature. In the discussion of the limb skeleton, for example, structural variations are related to differences in locomotor behavior.

The preface states that "the provisional interpretations . . . will probably not find universal agreement" and allusions are frequently made, if haltingly, to conflicting points of view. For example *Apidium* and *Moeripithecus* "may be" remains of condylarths; "some authorities" regard the dental formula of Parapithecus as 1.1.3.3. and "some paleontologists" have suggested that it is not a primate. Fortunately there is no elaborate reconstruction of an Anthropoid ancestral type based on these dubious fossils.

The dates of several references at the chapter ends are in error, but this is a minor point. The volume is the most useful compendium on comparative primate morphology that is presently available.

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Das Menschenbild der Biologie: Ergebnisse und Probleme der naturwissenschaftlichen Anthropologie (Biological Portrait of Man: Results and Problem of Anthropology as a Natural Science). By ILSE SCHWIDETZKY. With 81 illustrations. Gustav Fischer Verlag, Stuttgart, 1959. viii + 218 pp. DM 24.

It is a common experience that smaller textbooks in anthropology are apt to be disappointing, mainly because of their superficiality or incompleteness. In authropology, problems are so complex and are changing so rapidly that any attempt at completeness inevitably must lead to works of encyclopaedic dimensions. The preface of the present book indicates that the author is fully aware of the difficulties that go with writing a general work in a field such as human biology. Indeed, it becomes more and more difficult for one person to master all the sub-branches equally thoroughly.

The purpose of the present work is remarkably well summarized by the title itself: to draw a "Menschenbild der Biologie," a biological portrait of man. With this "Menschenbild" the author aims to compensate for the "Menschenbilder" drawn by philosophers and theologians. The sub-title excludes any possible misinterpretation of the meaning of the term "anthropology."

Schwidetzky presents a general, condensed survey of the problems, data and theories of the various fields of modern human biology. She sketches the place of man in nature (chap. I), discusses the problems and the present knowledge of human genetics as well as the influences of the environment (chap. II & III). Growth and constitution problems are treated, including a discussion of the recent modifications man has undergone (chap. IV). Chapter V deals with the origin of man, with a survey of the various fossil findings. Races and race system are discussed in chaper VI, while chapter VII is concerned with the factors determining evolution (Kräfte der Entwicklung)—a section including problems which in Anglo-Saxon works would be brought together under the heading "population genetics." Finally, there is a survey of sociobiological problems (Soziale Bevölkerungsdynamik) and a discussion of the phenomenon of culture as a product of organic beings (chap. VIII & IX). Although all this has been treated only in some 200 pages, Schwidetzky succeeds in providing a remarkable synthesis of anthropological facts and theory. The discussion of the nature-nurture relation-

ship or the interaction between "Siebung" and "Auslese" is excellent. As to the concept of race, we find a thorough presentation of the morphological and genetic aspects.

Striving for a comprehensive biological view of man, the author discusses morphological as well as physiological and psychological phenomena characterizing human populations and their evolution.

Among the best parts of the book is the section on social biology, a field in which the author herself has made numerous investigations.

Very little is found on biochemical genetics, and on hormones and behavior. The statistically minded anthropologists may also miss statistical formulae in the section on population genetics. Their omission, however, makes the text more readable. This book will be of great value to specialists in the social sciences and the humanities while, at the same time, serving as a stimulating guide-book to students of human biology.

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Heredity and Human Nature. By DAVID C. RIFE. 265 pp. Vantage Press, New York. $4.50.

This is a book about human genetics by a human geneticist, one long experienced in the study of normal human differences. It begins with an exposition of simple inheritance, using the very suitable non-Mendelizing M-N alleles, and it ends with some reflections on heredity and democracy, and a plea for open-mindedness on the hereditary nature of the more elusive human differences.

To the good is the author's use of his own data on isolate differences, including the atypical (but typically Welsh) ABO gene frequencies in the Joneses, and serological and anthropometric comparisons of Catholics, Jews, Lutherans and Presbyterians at Ohio State University. Twin data and twin comparisons including the coefficient of inheritability are also treated, though the coefficient itself is uncritically used.

To the bad, *Heredity and Human Nature* was born obsolete, being confined (except for a footnote) to 24 chromosomal pairs and no mosaics. The adaptive nature of human polymorphism is not treated, though the principles set forth by Lerner are recognized. Granted that Dr. Rife

is a geneticist and not a psychometrist, he has more faith in the Stanford-Binet than he should.

One wonders why the author chose the Vantage Press for they did not do well by him, especially as regards the bibliography, with references to "C. E. Coons," "W. M. Korgan," "C. A. Allison" and Wiener's "Blood Groups and Transfusion," Mendel's article in the Journal of Heredity for 1866, and Rife's own article in the "English Quarterly." The 24 tables are reproduced from photostats of typed originals, presumably using a 1906 Oliver, yet the headings are typeset! Most publishers, moreover, would have redrawn the nub-pen illustrations, including one picturing "phenylpyruvil acid."

The professional may want a copy of this book for the many unusual features contained, including comparative palm-print statistics. He will hesitate to approve it for student use.

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INDEX TO VOLUME 32

Age changes in—height and weight, 331-41; obesity, 348-57; subcutaneous fat, 239-48
Angel, J. L., 342-65
Archeology, of Iroquois ossuaries, 249-54
Australian aborigines, body heat conservation in, 4
Autopsy, demographic aspects of cases of, 185-96

Bailey, M. J., 274-87
Baker, P. T., 3-15
Blood groups—ABO and resistance to diseases, 24-5; ABO in West Africa, 19-20; natural selection in respect to, 17-27
Body build, 209-13—adaptations, 288-313; and climate, 3-15; and mortality, 322-3; mate selection for, 11; of American Indians, 288-313
Body surface area, 6-15
Body temperature, 4, 8, 10, 13
Body volume, 370-6
Body weight (see Weight)
Brain weight and behavior, 366-9
Burch, G. E., 135-61

Carns, Marie L., 370-6
Chiang, Chin Long, 221-38
Churcher, C. S., 249-73
Climate—and body build, 3-15, 289-99; effects on human evolution, 3-15
Cranial index of Iroquois, 268-71
Culture, in determinants of human evolution, 3-15

Damon, Albert, 331-41
Demography, 207-9; of prehistoric Iroquois, 249-73
Diseases—and obesity, 344-7; infectious, and natural selection, 24-5, 30-5, 57-8

Eskimos, body build of, 300-2
Evolution, human, effects of climate-culture interaction on, 3-15

Fat, subcutaneous, in Polynesian children, 239-48 (see also Obesity)
Fingernail growth, 119-34
Fry, E. I., 239-48

Gene flow—calculation of, 21-2; of sickle cell gene, 202
Growth—and development, 203-4; changes in subcutaneous fat, 239-48; of dentition, 112-4; of fingernails, 119-34; of hair, 319-20

Haptoglobins, 56-7
Harris, C. W., 370-6
Health problems, non-communicable in households, 167-81
Height and weight, 320-5, 331-41
Hellebrandt, F. A., 370-6
Hemoglobins, abnormal, 46-7
Heterosis in man, 74-5
Hillman, R. W., 119-34
Hulse, F. S., 63-79

Indians, American, 249-73—physique of, 302-8
Irradiation and genetic damage, 89-108, 316-8
Isolation, 80-8

Kenyon, W. A., 249-73

Lasker, G. W., 1-2, 80-8, 205-7
Liba, Marie R., 370-6
Life-expectancy—in obesity, 342-4; sample variance of, 221-38
Livingstone, F. B., 17-27, 197-202

Malaret, German, 135-61
Malaria, natural resistance to, 42-4; 45, 46, 47, 48, 50-6
Maternal death rates, 274-87
McFarland, R. A., 331-41
McMahan, C. A., 185-96
Migration, 80-8 (see also Gene flow)
Motulsky, A. G., 28-62

Natural selection in man, 17-27, 29; calculation of effect, 22-3; climatic effects, 9-11
Negroes, American—fingernail growth in, 119-34; reaction to cold, 4, 13, 14; reaction to heat, 4
Newman, M. T., 288-313
Nutrition and physique in American aborigines, 302-8

Obesity, 342-65

Peru, anthropometric differences between towns in, 81-2
Plasticity, 63-79, 86-7; in polygenic characters, 70-1
Polymorphism, 23, 28, 68, 70
Polynesians, subcutaneous fat in, 239-48
Population—breeding, effective size of, 84-5, 201; estimate of prehistoric from remains, 259-63
Prediction, test of, 162-84
Pregnancy, fingernail growth during, 119-34
Primaquine sensitivity, 48-55

Puerto Ricans, fingernail growth in, 119-34
Pygmies, 9, 17

Quiroz, Antonio, 135-61

Radiation (see Irradiation)
Random genetic drift, 20-1, 84-7
Reynolds, E. L., 89-108

Schade, Maja L., 370-6
Selection, 64-5 (see also Natural selection)
Sex differences in—femur, 255; hip bone, 205-7
Sex ratio, and rate of growth of mother's fingernails, 128
Sheehe, P. R., 162-84
Sickle-cell gene, 44-6; in Liberia, 197-202
Skin, 213-4; pigmentation of, 72-3
Skinfolds of Polynesian children, 240-8
Starvation, 29-30
Stature—and body build, 298-9; class differences in, 12; of Americans, 331-41; of Iroquois, from remains, 263-6
Stoudt, H. W., 331-41

Thalassemia, 47-8
Transferrins, 57

Veins, motor reaction in human, 135-61

Weight—and height, 320-5, 331-41; at birth and fingernail growth of mother, 125-8; body, 67; brain, 366-9; ratio with surface area, 6-15
Witherspoon, Y. T., 366-9

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Edited by GABRIEL W. LASKER

The six essays were presented in a symposium at the 59th annual meeting of the AMERICAN ANTHROPOLOGICAL ASSOCIATION at Mexico City on December 28, 1959, and marking the close of the Darwin centennial year.

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CONTENTS

HEIGHTS AND WEIGHTS OF WHITE AMERICANS. *Howard W. Stoudt, Albert Damon, and Ross A. McFarland* 331

AGE CHANGE IN OBESITY. *J. Lawrence Angel*..... 342

BRAIN WEIGHT AND BEHAVIOR. *Y. T. Witherspoon* 366

SEGMENTAL VOLUME REDUCTION BY LOCALIZED VERSUS GENERALIZED EXERCISE. *Marie L. Carns, Maja L. Schade, Marie R. Liba, F. A. Hellebrandt and Chester W. Harris*..... 370

BOOK REVIEWS..... 377

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#137